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LIPPINCOTT'S NURSING MANUALS

ESSENTIALS *of* MEDICINE

A

TEXT-BOOK OF MEDICINE

FOR STUDENTS BEGINNING A MEDICAL COURSE, FOR NURSES, AND FOR ALL OTHERS INTERESTED IN THE CARE OF THE SICK

BY

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ILLUSTRATED BY THE AUTHOR

[NINTH EDITION REVISED]

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PREFACE TO THE NINTH EDITION

In preparing the ninth edition of the ESSENTIALS OF MEDICINE we have attempted to make a little clearer the paragraph subjects. On the other hand, careful study has been made to avoid conspicuous titles and subtitles such as mar most text books, and we are very much pleased with the success that the J. B. Lippincott Company has had in handling this rather difficult problem.

The success of this book has been due in no small degree to the fact that the description of the conditions to be studied were logically based on the anatomy and physiology which the nurse might well be expected to know, and to succeed in presenting the subjects in that manner means the use of the narrative style.

Too much paragraphing and too many paragraph titles tend more and more to reduce the material to a series of disjointed statements with little logical connection, and merely to be memorized.

In this new edition we have been able to incorporate some new material, as perusal of Chapter XXI will show.

For this edition we have provided a very detailed Table of Contents to facilitate the reader in finding subject matter not readily identified by the single words of an index. Such a table of contents also aids the reader to evaluate the importance of the various subjects and their relation to the material as a whole. This to the beginner in medicine is of considerable importance since at first all medical facts seem to have an almost uniform value, whereas on the contrary they may vary much in importance.

THE AUTHOR.

PREFACE TO EIGHTH EDITION

When the ESSENTIALS OF MEDICINE first appeared very few training schools in this country were in a position to offer their pupils preclinical courses introductory to the study of medical nursing. Since then, through the efforts of the National League of Nursing Education, working in coöperation with the Schools of Liberal Arts, many of these training schools now are departments of Universities on a par with the other units of these educational institutions, and the great majority of the others already have, or soon will have, educational standards practically equal to those of the university schools. The actual progress made in nursing education, however, has depended in no small degree on the quality of the text-books used, and the widespread adoption, since its first appearance, of this book encourages us to believe that its influence has not been unimportant. Since text-books should not only meet but should anticipate the increasing capacity of the pupils who are to use them, the attempt has, from the first, been made by successive revisions to keep this book abreast of the progress of nursing education. This year the book has been entirely rewritten and will, we hope, meet the needs of the pupil nurse of tomorrow as well as former editions met those of pupils now graduated.

As at its initial appearance, our chief aim now in revising this book has been to avoid the dangers and evils of unconsciously "boiling down" a text-book of general medicine, and we have tried to base the subject matter presented on that knowledge which the nurse shall have gained in her preclinical courses and on her personal experience in the wards. In this way we may hope to assist her to develop some critical judgment concerning those ideas of general medicine which she picks up in such abundance in the wards and which she must of necessity accept largely on authority.

Especial attention has been made, in the discussion of each

disease, to emphasize those of its medical aspects which demand a definite response in terms of special nursing procedures if these patients are to enjoy those benefits of medical progress to which they are entitled.

C. P. E.

PREFACE TO FIRST EDITION

Several years' experience as a teacher of medicine in a medical school and medical lecturer to nurses has convinced the writer that there is a great need for a book similar to the one which he now respectfully submits to the reader.

Many American medical students lack perspective in their medical studies. They do not learn the A, B, C of the disease first and then proceed to its more difficult study.

During their second year they are taught the pathology of a disease, including a discussion of the nature of the disease as a whole and its most difficult points. During the next year they hear much of its clinical chemistry and microscopy, and more of the theories concerning it. In the fourth year perhaps they see their first patient with that disease. They read up at once, often in a large system of medicine, all about its symptoms, course, clinical varieties, complications, sequelæ, and treatment. And so it is no wonder that if at the end of the fourth year, in a quiz, the instructor asks a very simple question about that disease, they look confused. Ask about some difficult and worthless theory and they can talk at length. If he demands a definition of that disease in twenty words for instance, they look dazed. Ask for its four most important symptoms and the four they give will often be the disputed, the accidental, or the rare ones. They have not learned to separate the important from the unimportant. Our nurses often know a great deal in a general, indefinite, inaccurate way. They seldom have a clear sharp mental picture of the elements of a subject.

It is for these two groups of readers especially that this book was written, in the hope that from it those beginning

the study of Medicine and nurses may gain as their first impression a clear if limited idea of a subject.

This book may be attractive to the general reader. It will aid him to understand more clearly the medical problems of the day, to appreciate more highly a well trained practitioner, and to coöperate better with his doctor. In this manner it will fulfill the purpose of its author, and likewise will serve the need of the lay-reader far better than if he attempts to use it as a family physician, replete with information concerning the diagnosis and treatment of his ailments, an office it is not intended to fill.

CHARLES P. EMERSON

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Essentials of Medicine

CHAPTER I

Physical Signs and Symptoms

The trained nurse is no longer a mere helper in the sick-room, but a professional associate of the physician, a specialist in her own line. She is entrusted with the continuous care of the patient and is held responsible not only for the carrying out of the doctor's orders, most of which an untrained woman could do, but especially for the correct observation and recording of the important features of the case. One of the main results of her education is the ability to recognize promptly each disease's symptoms and signs, especially its danger signals, to assist in the therapy, and to minister to the comfort of the patient.

Symptoms are those evidences of disease which only the patient has, his aches and pains, for example. These are subjective. **Physical signs,** on the other hand, as the swelling of parts of the body, the sounds from the heart and lungs, are objective. These the examiner, unassisted by the patient, can discover and study; about these he can form a personal judgment. There are also other evidences of disease which are objective, but which a patient might, intentionally or unintentionally, produce, as cough, vomiting, etc. According to the case presenting them these are considered sometimes symptoms, sometimes physical signs. For example, in a case of pleurisy the pleural pain on breathing is a symptom (we should know nothing about it unless the patient told us); the restrained respiratory movements of the affected side of the chest some would call a symptom, since the patient could fake these if he wished, while others would call them a physical sign because they can see them; but the audible friction rub—that is a physical sign. Over that the patient has

no control. To record the symptoms accurately and to observe and record intelligently those signs which it is within the province of the nurse to observe, these are two of the best criteria of a nurse's education and training.

It is of special importance that the **mental condition** of the patient be watched. While marked abnormalities are easily recognized, one may easily fail to detect their first traces, which indicate the time to begin active therapy or to guard the patient or his friends. A correct evaluation of these symptoms is very important also in determining what the nurse's attitude towards the patient should be, whether one of sympathy, of encouragement, or of indifference; and whether she should warn the doctor or should use all means at her disposal to meet the immediate needs of the case. The experienced nurse will be prompt to note **the keen, attentive, interested look** of the **neurasthenic** who fears that one may miss or underestimate some details of his condition; **the worried, frightened, self-depreciative and depressed look** of the **psychasthenic**; the **incongruous look** of the patient with **true hysteria** who relates as if of little importance details of her paralysis or blindness, symptoms which would terrify a more normal person; **the depressed, hopeless look** of the **depressed patient,** as well as any sudden restlessness on his part, which may mean suicide when her back is turned; the inattentive, excited look of the case of mild mania; the fatuous expression of the paretic; etc.

Degrees of mental alertness are conditions which it is important to observe: **apathy,** or mere comfortable indifference, well illustrated in nearly all typhoid patients and sometimes marking a serious development of the case; the **twilight condition,** in which the patient is half-awake, and often delirious when quiet, but easy to arouse; **somnolence,** which renders the patient abnormally sleepy, and which is sometimes the first sign of oncoming diabetic or uræmic coma; **stupor,** from which the patient can always be aroused but only with difficulty, as in the case of one drunk; and **coma,** from which he cannot be aroused.

Conditions of mental confusion should be observed. In various mental states the patient is not "orientiert" as to time, place, persons, etc. That is, he is not sure where he is, or how he came there; he is doubtful as to the time of day, or the day of the week, etc.

Unusual loquaciousness, mental exaltation or the reverse—depression of spirits—are often important signs. Any indication of ideas of persecution, a distrustful attitude, and any "delirium" in a patient without fever should put the nurse on her guard.

While all the mental symptoms mentioned above may be evidences of actual mental disease, yet all may be symptoms also of acute infectious diseases.

Symptoms are really **poor data in judging of the nature or the severity of a disease,** and yet it is symptoms that trouble the patient, and, no matter what his condition may be, when they disappear he calls himself well. Herein lies the success of quacks. They relieve symptoms, and their patients are satisfied. And herein lies the failure of many of the best trained doctors. They concentrate their attention on the disease but neglect to make a patient comfortable. To relieve symptoms and to cure disease are two separate problems, both important. While **physical signs** are the province of the **doctor, symptoms** are in a peculiar way **that of the nurse,** and she should apply herself to learning those numerous little "tricks" of her profession by which they may be relieved, remembering that no symptom is too trivial to be disregarded, and that the skilful application of simple nursing methods often changes a complaining sufferer into a contented and fairly comfortable patient.

The public, long educated by the advertisements of various proprietary medicines—advertisements that emphasize abnormal feelings—has been slow to believe that there can be **serious disease without symptoms,** and yet this is true of several serious diseases for most of their course and of a larger number of diseases during some part of it, especially the early part, while they are still curable. The small cancer that will

finally kill the patient is often unobserved for a long time. Many a man discovers when he applies for life insurance that he has heart disease or advanced Bright's disease, which thus far has been entirely without symptoms, but which later will be only too evident. Some man who died from accident is found at autopsy to have had an aneurism which he never suspected or a gastric ulcer almost ready to perforate the stomach wall. Another man, who never knew that he had a serious heart trouble, falls dead on the street. Many more illustrations might be given, but these are enough to show how hopelessly at variance are our feelings and our condition.

Many serious diseases make their presence known to the patient chiefly **through disturbances of organs other than the ones diseased,** disturbances some of which can be cured without relieving the primary disease, and others only when this has been accomplished. Here is a patient who sits upright in bed in his effort to breathe. He has a distressing cough and expectorates much sputum, and also blood in considerable amounts. Examine his lungs, and you will find severe congestion and acute bronchitis. He is sure that he has a serious lung trouble. What he really has is bronchitis which would be trivial for a more normal person, but which is serious for him since it is indirectly due to a heart trouble. We may "cure" his "lung trouble" without his discovering his heart trouble, but, if we relieve his heart condition, his "bronchitis" will clear up in a few days. Here is a patient with "rheumatism of the feet"; they are swollen and sore. Or, he may have trouble at the opposite extremity of his body—a terrible headache, and hæmorrhages into his eyeballs. Or, it may be his stomach, which distresses him, and any one seeing his attacks of vomiting will believe that there lies the disease. But neither his feet nor his head nor his stomach is diseased; his real disease is in the kidneys, organs which have not given a single clear symptom.

Sometimes the **primary trouble is much less serious than those which it causes.** This patient has chronic laryngitis and bronchitis, but it may be an unsuspected suppuration in

the nose which originally caused these troubles, and which now keeps them alive. Here is a dying man. He has had repeated attacks of that most terrible disease, acute endocarditis, which have injured his heart, each more than its immediate predecessor. Now he is in a pitiful condition and cannot live long. The man in the next bed has had repeated attacks of acute arthritis and has become a helpless cripple. The primary trouble in both these cases may have been a simple tonsillitis which they do not know they have had. Could the tonsils have been removed months or years ago, the chances are that these men would now be free from the diseases that afflict them.

In a much larger group of cases the **organ which gives rise to the symptoms is not diseased.** In each ten **(some say twenty)** cases with symptoms of stomach trouble not more than one will have a stomach disease. To treat the stomachs of the other nine (perhaps nineteen) would be like pouring water on the fire department building because that is where the fire bell is ringing. So the stomach frequently gives the signal of trouble elsewhere in the body. Some persons have the most distressing attacks of asthma—attacks which may lead to permanent and serious lung injuries—and yet these attacks are sometimes only signals indicating trouble in the nose.

Finally, we must in this connection speak of **referred pains,** severe pains distant from the seat of the disease. In gall-stone colic much, even all, of the pain may be in the shoulder; in acute inflammation of the gall-bladder the pain may all be also in the left side, a foot away from that organ; in pelvic trouble the pain is sometimes in the back of the head; in incipient hip disease the pain may be in the knee; in heart disease all the pain there is may be down the left arm, etc.

The illustrations given above show how easily one may be deceived if he depends entirely on symptoms. If one searches for signs with seeing eye, he will in practically all such cases as those cited locate the real trouble. Nevertheless, these symptoms are important to the nurse, since symptoms,

wherever they are and whatever they mean, **ought to be relieved.** If morphia is necessary for severe referred pain, there will be greater relief if the injection is at the seat of referred pain and not always in the upper arm, for where the pain is felt, there it is. There are anatomical reasons for these referred pains, and local treatment with hot water bottles, poultices, etc., at the seat of pain not only relieves that pain but does help the original trouble, however distant it is. Finally, pain is an unpleasant sensation, nothing more, and is never imagined. Imagination may be its cause, but the pain thus produced hurts just as truly as pain produced by a real disease. Pain is a phenomenon of consciousness, and is always real, even when felt in a dream. If the patient is too unconscious to feel pain, there simply is none, no matter how badly the person's body may be injured. If the patient has it, the nurse must try to understand it and to relieve it, whatever its cause.

CHAPTER II

General Attitude and Appearance of Patients

Much information valuable in diagnosis is offered by the patient's **physical positions,** that is, by his attitude. Those attitudes which he assumes voluntarily are called **active.** If he is so weak or helpless that he lies passively in whatever position gravity or accident may determine, his attitude is described as **passive.** Active attitudes which the patient voluntarily or, as in the case of joints, unconsciously assumes in order to get relief are called **forced attitudes.**

The attitude and expression of the psychasthenic often express anxiety; that of the mildly maniacal, activity; that of the case of exophthalmic goitre, fear; that of the depressed case, dejection; that of the patient with typhoid fever, drowsiness. The appearance of lethargy commonly marks patients with epidemic encephalitis, who will sit still for hours and, though hungry, not touch food placed before them.

The patient with true angina quickly immobilizes himself—he can scarcely do otherwise; one with hysterical cardalgia gets comfort from movement. A patient with cardiac incompetence, in order that he may breathe as easily as possible, will prop himself up in bed, and, the greater his distress, the more erect will he sit. If the dyspnœa is very severe, he may even lean forward (orthopnœa), propping his arms against the bed, or, if in an armchair, bearing down with his hands on its arms in order to raise his shoulders. When he is in this position his diaphragm takes the lowest possible position, and the capacity of his thorax is maxinal, which is always important for his comfort, particularly if there is fluid in the chest or abdomen.

Some patients with goitres under the sternum or with tumors in the chest cannot lie down but sleep in a chair with the head bent forward and supported on the bed or another

chair. When they lie horizontal, the tumor falls back, pressing against the windpipe or lung.

Some patients can **lie comfortably only on one side.** Those with fluid in only one pleural cavity usually lie with the heavy side down in order that the weight of the fluid may not oppress the better lung. Patients with acute pleurisy lie with the affected side uppermost in order that the weight of the intrathoracic organs may not increase the friction between the two inflamed surfaces. Those with every breath painful because of an acute rheumatism of the joints between ribs and vertebræ usually so lie that the weight of the body keeps these joints as quiet as possible. Cases with cardiac dilatation usually prefer to lie on the left side, since then the heavy heart falls against the left wall of the chest. If they lie on the right side, it will tend to fall against the right lung. Patients with acute bronchitis, or acute pneumonia, will lie fairly flat and often comfortable even though they are blue and breathless.

The attitudes assumed by **patients with abdominal pain** may suggest the diagnosis. If they get relief by pressing against their abdomen, by doubling up or by lying on their face, especially if across a hard pillow, their attitude suggests a painful gastric or intestinal peristalsis, popularly called "colic," which the pressure partly stops. Those on the contrary with inflammatory conditions within the abdomen, e.g., an acute appendicitis, gall-bladder infection, or peritonitis, avoid all pressure. They prefer to lie on the back with knees flexed, in order both to hold the bed clothes away from the abdomen and to lessen the tension of the infected tissues. Patients with painful stomachs prefer to lie on the right side; those with painful gall-bladders, on the left; etc.

The forced and often odd appearing attitudes assumed by patients who have painful muscle spasm, as stiff neck, lumbago, sciatica, are merely attempts, often vain, at greatest ease. Patients with any form of acute arthritis hold the inflamed joints semiflexed, because that position allows the greatest capacity to the synovial sacs full of fluid. The

hemiplegic patient will assume that position which gives his good side every advantage. The hysterical attitudes are particularly important in diagnosis since they are unconsciously assumed by the patient and determined by some dominant wish or fear.

Finally, **the general appearance of the patient may suggest his disease.** The sum total of evidence of any one disease afforded by the face is called the **facies** of this disease; when all signs are included, it is called the **syndrome** of disease. **Anæmic persons** are not always pale. Their cheeks may be flushed, their lips and tongue pale. The greenish tint of the pale skin of a case of chlorosis is suggestive, but her very pale lips are much more so. The facies of pernicious anæmia includes a definite pigmentation of the skin, similar to sunburn, which may deceive one, but also marked paleness of the mucous membranes of lips, tongue, and mouth. Some persons who are very pale have no anæmia at all, merely a limitation of blood in the external capillaries. This is clearly seen in aortic insufficiency and nephritis.

The sick looking patients, victims of diseases of long duration which cause great prostration, are thin, pale, tired, haggard, their eyes are sunken and their cheek bones prominent. The person with advanced cancer or with long continued suppurations looks thin, pale and tired **(the cachectic facies).** The best example of this is the **Hippocratic facies** of the person dying of peritonitis. Hippocrates' description is as follows: "A sharp nose, hollow eyes, collapsed temples; the ears cold, contracted and their lobes turned out; the skin about the forehead being rough, distended and parched, the color of the whole face being green, black, livid or lead-colored." On the other hand, patients dying from other diseases may not look very ill.

The child with **marked nasal obstruction** looks stupid, heavy, apathetic. He responds slowly to questions and often is sullen and cross. His nose is rather pointed at its tip and has nostrils which are small, narrow, and pinched, and which retract during sleep with each inspiration. His mouth is

usually open. His lips are thick, the superior dental arch is narrow, the roof of the mouth is high, and the teeth as a rule are carious.

Patients with **hookworm disease** have an apathetic and listless expression, dilated pupils, and a very characteristic "dull," blank, almost "fishlike" expression (the "cadaveric stare").

The physiognomy of the patient with **myxœdema** betrays the slowness of his psychomotor activities.

The patient with marked **exophthalmic goitre** has a characteristic staring expression suggesting "crystallized fear," caused in part by the exophthalmos, but more by the retraction of the lids, which exposes the scleræ, the smoothness of the forehead, and the tension of all the facial muscles.

Patients with conditions producing **dyspnœa** usually lie propped up in bed, their faces cyanotic. (See page 116.) **The cardiac facies** of cases of long-standing mitral disease is characterized during periods of good compensation by suffusion of the skin, cyanosis, especially after exertion, of the lips and finger tips, dilation of the superficial veins, and yet by the absence of œdema. Patients with aortic insufficiency, on the other hand, look pale, anæmic, and thin.

The nephritic facies is characterized by marked paleness of the skin and mucosæ (arousing a suspicion of anæmia which may not be confirmed by the blood count), by puffiness of the lower lids at least, sometimes by puffiness of the whole face obliterating the wrinkles of expression, by blue scleræ, and often by the œdematous conjunctivæ.

The hepatic facies is sometimes unmistakable. The patient with liver disease is thin, especially in his face and neck, and his complexion is muddy or jaundiced. The eyes are sunken, the conjunctivæ watery, and the venules over the nose and cheeks distended. Nævi of various varieties (the stellate varices, the spider angiomata, and especially the mat nævi) often appear on the skin of the face but also on the neck and back.

Incipient **facial erysipelas** appears as a red area of skin, with a sharply defined swollen edge, which spreads from the bridge of the nose over the cheeks. Erysipelas may develop elsewhere on the face, but always shows the same clearly marked advancing edge.

Patients with **early pulmonary tuberculosis** may appear perfectly well, though in the afternoon they do look tired. The advanced case, however, is emaciated, and his lips as a rule, not always, are pale, while his cheeks have a deceptive hectic flush.

Acute lobar pneumonia gives its victims a more distinctive facies than does any other acute disease. A patient with this trouble usually lies on the affected side and breathes rapidly, the alæ nasi dilating with each inspiration and each breath accompanied by a short expiratory grunt. His cough is short and evidently painful; he is cyanotic but not dyspnœic; one or both cheeks are flushed; herpes is usually present on the lips or nose; his eyes are bright; their pupils are often unequal; and his expression is anxious.

Early in an attack of **typhoid fever** the patient's cheeks may be flushed and his eyes bright, but by the end of the first week his expression has become listless, and later it is dull and heavy. Then he lies quiet wishing only to be left alone, his mouth partly open, his tongue and gums dry and covered with sordes. Since the anæmia of typhoid fever develops slowly, his lips and cheeks may have good color even until the third week.

A patient with **diphtheria** looks, and is, much sicker than he feels. His face is pale, even ashen. He appears prostrated and is quiet unless dyspnœic because of laryngeal involvement. A patient with follicular tonsillitis, on the other hand, has a flushed face and appears very uncomfortable.

The patient with **malaria** usually appears comfortable but weak, rather drowsy, anæmic, and slightly jaundiced.

A patient with **yellow fever** may, even on the first day of his illness, present a characteristic facies which Guiteras described as follows: "The face is flushed, more so than in

any other acute infectious disease at so early a period. The eyes are very red, and there may be a slight tumefaction of the eyelids and lips. Even on this first day there is in connection with the injection of the superficial capillaries of the face and conjunctivæ a slight jaundice, the early appearance of which is undoubtedly the most characteristic feature of the facies of yellow fever."

A patient with a case of fully developed **mumps** needs no description, but very early in this disease, before the parotid swelling is pronounced, this condition is suggested by a slight swelling high in the neck, higher than that due to enlarged lymph nodes, so high that it lifts the lobule of the ear. This swelling when more pronounced overlies the ramus of the jaw just in front of the lobule of the ear, a fact which aids in distinguishing swellings of the parotid gland from cervical adenitis.

The **masklike expression in Parkinson's disease** is almost characteristic, even very early in cases of paralysis agitans. The features of the patients are expressionless and immobile, the eyebrows elevated, and the movements of the lips slow.

The **risus sardonicus of tetanus** is so called because the patient's mouth has a smiling expression while his eyes look sorrowful. This effect is due to the differing relative strengths of the various groups of facial muscles when all of them are in spasm.

CHAPTER III

Temperature, Respiration, Pulse and Blood Pressure

Temperature

The temperature of the internal organs is usually higher than that of the external air, and in health is practically constant. The heat of the body is generated in the muscles during their contractions, and in the large glands, where combustion is very active. From these warm organs the blood is constantly flowing to cooler parts of the body, and cooler blood is always flowing from the latter to them, while at the same time there is a continuous loss of heat from the skin and the lungs and in the secretions.

Though the external temperature varies during the year by about 120° F., the temperature inside the healthy body does not vary more than two degrees, because there is at the base of the brain a "heat-regulating centre," which controls matters with wonderful accuracy. When the outer air becomes cooler, the blood vessels in the skin are made to contract, and so on the surface of the body there is less blood to cool off. The cooled skin stimulates us to make our muscles contract oftener and with greater force. When we are cold and do not contract them voluntarily, they contract involuntarily, and we "shiver" and our teeth chatter. The sensation of cold leads us to put on warmer clothes, to eat more food, and to drink hot fluids. By the first act we diminish heat loss, by the second we increase oxidization, and by the third we add heat directly to our body. When there is a tendency for the body temperature to rise, the skin vessels dilate, so that more blood is where it can cool rapidly, we perspire more, and the evaporation of fluid from the skin cools us considerably. We exercise less, dress more lightly, eat less, take cold drinks, each of which practices either lessens the heat production or increases its loss.

The body temperature is fairly constant; the skin temperature varies greatly. The old method of determining body

temperature was to put the hand on the skin and decide whether or not the patient felt feverish. This method is roughly accurate, provided the hand is placed on a part of the skin protected at the time by the clothes, so that there is just then no great heat loss, and provided the patient is not in a chill or sweat. In the case of a chill the skin may actually be colder than normal, and yet the blood temperature high; in the case of a sweat the skin may be warmer than normal, and yet the blood temperature normal or a little below normal. **The blood or body temperature** is now determined by a **thermometer** placed in the mouth, the axilla, or the rectum. Each method has its advantages.

In America the temperature is generally taken in the mouth. The thermometer is placed under the tongue, the lips are tightly closed and kept so, and the thermometer is not removed until its mercury reaches a constant point. How long this will take depends on the construction of the thermometer, but, even if the most sensitive thermometer is used, at least two minutes are necessary, since the mouth, after it is closed, requires at least that time to warm up to the blood temperature. In other words, while there are "one-minute thermometers," if tested in water, there are no "one-minute patients," and so one gains little from the use of these sensitive instruments. With very ill patients at all toxic the temperature should not be taken by mouth because, while they try to keep the lips tightly closed, they do not always succeed. Another disadvantage of mouth temperatures is the danger of infecting the patient by a thermometer not thoroughly cleaned which has just been used by a person with a communicable disease.

The **axillary temperature** is easily and more hygienically obtained, but to take it requires a much longer time, at least fifteen minutes. The nurse must be sure that the arm under which she places the thermometer is held close to the body. One of the best ways to accomplish this is to turn the patient, after the thermometer is in place, over on that side.

For very ill patients the **rectal temperature** is preferable,

as nothing is required of the patient. The same precautions against infection must be observed as in the case of the mouth, with the added precaution that before the thermometer is inserted in the rectum it should be well oiled with vaseline. The nurse must also be sure, even if this requires an enema, that the rectum contains no fæcal masses, however small, since heat is always being generated in fæces, as any one can infer who looks at a manure pile on a cold day. The bacteria of the colon are continuously decomposing, that is, are "burning

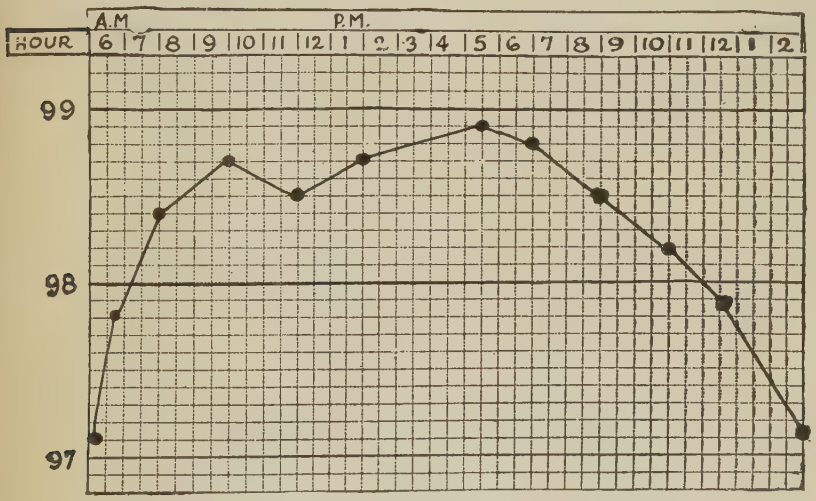


Fig. 1.—Normal hourly temperature curve for one day.

up," some of the organic matter of the stools, with the result that the temperature of the fæcal mass in the rectum is that of the intestinal wall plus the degree or so added by this local combustion.

Of these three temperatures, each taken with the greatest care, the rectal will be a trifle the highest, about half a degree, and the axillary temperature a trifle the lowest. In fever cases the temperature is taken once every two or four hours until it is practically normal, after which it is taken only at 8 A. M. and 4 P. M. The normal temperature is about

98.7° F. The readings should always be charted, for the shape of the platted temperature curve is instructive.

In order to interpret correctly pathological **temperature curves** one must first clearly understand that of a normal person (Fig. 1). Many students seem to think that the little mark on the scale between 98° and 99° F. indicates a constant normal, rather than the mean normal. The temperature of a normal person during the twenty-four hours may vary over a range of almost 2° F. At 4.00 A. M., when the temperature is lowest, it may be as low as 97.1° F. It then slowly rises until, between 9.00 and 10.00 A.M., it reaches about 98.7° F. During the next four hours it often drops about 0.2° F., after which it rises slowly, reaching its maximum, 98.9° F., between 5.00 and 8.00 P. M. The temperature then slowly falls until 4.00 A. M. the next morning. The temperature of night workers gives the reverse curve. The strictly normal temperature curve does not touch 99° F. An early morning temperature cannot be called **subnormal** unless it is below 97.1° F. In the afternoon, however, 98° F. would be subnormal.

Hyperpyrexia means a temperature over 105° F., or, better, over 107° F. If there is slight fever, as in latent tuberculosis, the temperature may rise to 99° F. or more for a few hours in the late afternoon, during the rest of the day being within, or even below, normal limits. In all fevers there is a tendency for the curve to maintain, but on a higher level, the form of a normal curve with the normal variations much exaggerated. The fever curve, however, is not always normal in form, since its maximum may come at noon, in the forenoon, or late at night.

Fever is usually a reaction against infection, but it may be the effect of the absorption of blood after an internal hæmorrhage; of some drug, as atropin; of increased tissue metabolism, as in a case of exophthalmic goitre; of disturbed heat regulation, as in sunstroke; or of some disturbance of the thermoregulator centre, as in cerebral hæmorrhage, brain tumor, and other cerebral lesions. Nervousness, in the sense

of emotional distress, is said not to raise the temperature, but specialists in pediatrics disagree.

While the toxins of germs may be called the "cause" of the elevation of temperature, the latter is really a "reaction." Fever in a case of infection seems to be a protective measure on the part of the body, a means to an end, and that end is a fight against the germ. The body would seem to have more resistance against germs at a temperature above normal, and so a fever is rather more a part of the defense than a direct result of the toxin. We emphasize this point because so many persons think the fever an evil and try to lower it with drugs. They can do this easily but with no benefit—perhaps always with injury—to the patient. We like to see the temperature fall after a cold bath, but only when we are sure that the total result of the bath is beneficial. Of course the temperature may be dangerously high, and then it must be reduced by any means possible. A sunstroke, for instance, the result of the breakdown of the thermoregulator centre, and with a fever of from 109° to 112° F., is treated by a continuous cold bath, spongebaths of ice water, ice packs, ice water enemata, etc. Some of these cases, however, fall as if struck down and die at once.

It may be said, in general, that the **height of temperature** is really **no index of the severity** of the case. The highest temperatures occur in the least serious fevers, such as relapsing fever and malaria, while in the rapidly fatal infections there may be no rise of temperature at all. In the latter cases it seems as if the body were unable to make a febrile defense against the infection.

The type of fever is judged by the highest and lowest temperatures of one day. The fever is said to be **constant or continuous** when these do not differ by more than 2° F.; **remittent,** when they differ by not less than 3° F. but without the curve's touching normal; **intermittent,** when, after a drop of 3° F., the low temperature is at or below normal.

The temperature may **rise suddenly, usually with a chill,** as in malaria, pneumonia, scarlet fever, and most of the fevers

that occur during childhood; or slowly, that is, during two or more days, as in typhoid. The best examples of constant fever are lobar pneumonia (see page 421) and typhoid fever at the end of the first week and at the beginning of the second (see page 404). The temperature may fall suddenly, usually with a sweat, as in malaria and pneumonia; or slowly, during several days. If the temperature falls from a high point to normal within twelve hours, the fall is called a **crisis** (see page 418); if within twenty-four hours, it is called a **prolonged crisis** (see page 423); if the fall requires more than twenty-four hours, the temperature is said to fall by **lysis** (see page 422).

Respiration

The nurse should **count the respirations** while the patient is unaware of what she is doing, for if he is conscious of her action the rate will almost certainly change. The easiest way is to pretend to be counting the pulse. The respirations are counted for at least half a minute, better for a whole minute. **The normal rate** for an adult is from 16 to 24 per minute, for a child about 20, for an infant about 44. When counting the respiratory movements the nurse should note also their type, rhythm and depth, the position which the patient takes as that of greatest comfort, any unusual effort that he makes in order to breathe, any evidence that breathing causes pain, and the presence or absence of cyanosis.

A person **inhales** by increasing the capacity of the chest. This is done by raising the ribs (costal breathing) or by lowering the diaphragm (abdominal breathing) or by both. He **exhales** by allowing, or forcing, the chest wall to fall back to its previous position and the diaphragm to return to its position of rest. Women breathe mostly with the upper chest, in later life their respiration becomes more and more abdominal. In men the respiration is from the first abdominal, that is, diaphragmatic, in type. **Any reversal of type** demands explanation. For illustration, the respiratory movements are **entirely costal** (or almost entirely) if for any reason the movements of the diaphragm are limited, as by diaphragmatic

pleurisy or paralysis of the diaphragm, by the weight of fluid in the pericardium above, or by pressure from below which pushes the diaphragm up, as that due to fluid in the abdomen, gas in the bowels, an abdominal tumor, or general peritonitis (a "quiet" abdomen strongly suggests tuberculous peritonitis). **Very exaggerated costal respiration** is seen best in hysteria. The breathing is **abdominal** only if for any reason the costal movements are limited, as by rheumatism of the costovertebral joints, fluid in both pleural sacs, extensive pneumonia of both lungs, or any condition which weakens the chest wall movements (as some forms of paralysis) or makes them painful.

Shallow respiration is the rule when for any reason the lungs do not expand well. They may have lost their elasticity, as in emphysema or because inflammations have deposited too much scar tissue in them, or the respiratory movements may be painful, as from pleurisy, neuralgia, or acute arthritis of the rib joints, or may be greatly limited, as from chronic arthritis of the costovertebral joints. Some patients move the ribs on one side of the chest more than those on the other. This may help us to tell on which side the pneumonia, pleurisy, or tuberculosis is, for that side will expand less. The slight lagging in the expansion of one apex is especially important since this often indicates a previously unsuspected tuberculosis of that upper lobe.

Rapid respiration (polypnœa or tachypnœa) may be due to various causes. It is physiological for normal young children, and for adults after exercise; in the latter case the breaths are also deep. Some nervous persons breathe even sixty times a minute; the respirations are so full, easy, and painless that we cannot say that the patient has **dyspnœa,** for that word implies an effort to breathe, but they certainly have polypnœa. Other nervous patients breathe slower than is normal, these have oligopnœa. If a person every minute or so takes a long, sighing breath, there is good evidence that he is neurotic.

Polypnœa (not dyspnœa) is just as much a part of the

fever syndrome as is the rise of temperature, and the rate of respiration increases five per minute with each degree in the rise of temperature. In the acute pulmonary conditions, in which there is no pain on respiration, as acute lobar pneumonia, the rate of increase is higher, sometimes even ten for each degree of temperature. In all fever cases this polypnœa depends on a special stimulation, by the toxin of the disease, of the respiratory centre located at the base of the brain. This is true of the febrile pulmonary conditions also. When a case of pneumonia recovers by crisis the temperature, the pulse rate, and the respiratory rate all fall rapidly and reach normal hours, sometimes days, before the inflammation of the lungs has even begun to clear up. Polypnœa may be the result also of the effects of any pulmonary disease which lessens the breathing capacity of the lungs, as when this organ contains much scar tissue. These patients may breathe normally while sitting quiet, will breathe faster (i.e. will develop polypnœa) when they walk fast, and will become dyspnœic when they move still faster.

Slow breathing (oligopnœa) is best exemplified by the toxic effects of opium, chloral, chloroform, aconite, or antimony; it is a feature of coma, collapse and shock due to any cause; it is a symptom of increased intracranial pressure, as from cerebral tumor, apoplexy, or meningitis; it may be due to hysteria. Finally, in bronchial asthma the oligopnœa is pronounced, but here there is dyspnœa also; each slow breath requires an effort.

By **dyspnœa** is meant difficult (labored) respiration. The respiration of a person in dyspnœa may be either fast or slow, regular or irregular in rhythm, and shallow or deep; but to deserve the term dyspnœa the breaths must be manifestly difficult, though not necessarily uncomfortable; in fact, in a mild case the patient may be unconscious that he is in dyspnœa. The proof of dyspnœa is that the accessory muscles of respiration can be seen in action; that is, one can see that the neck muscles contract to assist in respiration, lifting the clavicle at each breath, and usually that the alæ nasi dilate

with each inspiration. In severe dyspnœa, always uncomfortable, the patient's expression is anxious, the pupils dilated, the skin moist, the tongue and lips dry, the mouth open, and, usually, the skin cyanotic. A very severe dyspnœa is practically always also an orthopnœa; that is, the patient cannot lie down but must always sit up in order that the diaphragm may drop to a lower level than when he is horizontal. This of course increases the capacity of his chest. If the orthopnœa is extreme, the patient will even raise himself on his arms in order to lift his shoulders, thus increasing still more the capacity of his chest. **A dyspnœa may be constant or paroxysmal.** It may affect **inspiration only** (inspiratory dyspnœa), with expiration fairly normal, as in cases of pressure against the windpipe; or it may affect **expiration only** (expiratory dyspnœa), inspiration normal, as in asthma. **In mixed dyspnœa,** clearly seen after violent exercise, there is difficulty with both inspiration and expiration.

The rate of respiration in dyspnœa and also the amount of effort during respiration are controlled by a nerve centre which is stimulated either by too much carbon dioxide in the blood or by some poison circulating in it. The cause of dyspnœa, too much carbon dioxide in the blood, may therefore lie in the lungs, in the circulation, or in the outside air. Dyspnœa which is strictly pulmonary in origin **(pulmonary dyspnœa)** may be the result of a marked diminution of the respiratory surface of the lung, which may have been destroyed, as by hypertrophic emphysema, tuberculosis, or any inflammation or tumor which destroys lung tissue. It may be the result of any condition which temporarily fills up the air cells, as pneumonia or œdema. It may be due to any condition which causes the lungs to collapse, as the presence of fluid in the pleural cavity, in the pericardium, etc.

Cardiac dyspnœa, due to the inability of the heart to maintain satisfactory pulmonary circulation, as a result of which the carbon dioxide is not sufficiently removed from the blood, may be due to any trouble which weakens the heart's action. It differs somewhat from all other forms of dyspnœa because

of its greater tendency when severe to become an orthopnœa and to allow the production of a pulmonary œdema. That form of cardiac dyspnœa which resembles asthma (cardiac asthma) is especially distressing.

The **dyspnœa due to mechanical obstruction** of the upper air passage is inspiratory in type, each inspiration being slow and deep. When it is severe, the intercostal spaces and the wall of the upper abdomen seem sucked in by each inspiration, since each breath increases the capacity of the chest much faster than the air, because of the obstruction, can rush into the lungs to fill the potential vacuum thus produced. Stridor is always present. This form of dyspnœa may be due to huge tonsils, a large adenoid, or a retropharyngeal abscess; to closure of the larynx from diphtheria, œdema, or inflammation; to spasm or paralysis of the laryngeal muscles; to scares which by contracting cause stenosis of the larynx, trachea, or a primary bronchus; to foreign bodies or tumors in the windpipe; or to pressure against the trachea by an aneurism, an enlarged thyroid, or a tumor. In the case of young children, some say it may be caused by the thymus.

Expiratory dyspnœa may be due to partial closure of the smaller bronchi, caused by spasm, as in bronchial asthma, or by the presence there of an exudate, as in diffuse bronchial pneumonia.

Dyspnœa due to any mechanical restriction of the movements of the chest wall is mixed in type and usually is unilateral, partly because the restriction generally affects one side only, and partly because the normal side will make excessive movements in order to compensate for the lack of movement on the other side (compensatory overaction). Such dyspnœa is seen temporarily in case of acute pleurisy or of acute arthritis of the chondrovertebral joints on one side, but when permanent it is the result of some chronic condition: of arthritis which fixes these joints on one side, of paralysis of the respiratory movements, of a large tumor in one half of the chest, of collections of gas or fluid in the pleura, or of

some trouble below, in the abdomen, which pushes the diaphragm up into the chest.

Toxic dyspnœa, that is, dyspnœa due to the stimulation of a poison in the blood stream, includes the dyspnœa due to fatigue and that due to the acute fevers. It is a feature of many serious diseases, including Bright's disease with uræmia (some of these cases are called "renal" asthma because they so closely resemble true asthma). In diabetes mellitus with oncoming coma there is diabetic dyspnœa, Kussmaul's air hunger, a dyspnœa without cyanosis, with respiratory movements which are very deep but not necessarily hurried, and which require considerable effort though they cause little, if any, distress.

The **dyspnœa** experienced **at great altitudes** or in impure air is due not to bodily troubles but to the atmosphere itself. That which develops in **anæmia** is due to the lack of hæmoglobin, so important in respiration.

In conclusion, **dyspnœa following relatively slight exertion** is an important sign of latent phthisis, of slight cardiac disease, of which it may be the first symptom, of chronic bronchitis, of emphysema, of anæmia and, of course, of obesity.

Respiratory arrhythmia, or irregularity in the respiratory rhythm, if temporary, is due usually to psychical influences. Catching the breath, "holding the breath," periods of rather deep, restrained breathing, etc., are signs of mental tension. An occasional, long, sighing breath, recurring at rather regular intervals, is fairly characteristic of hysteria. An irregular jerkiness of respiration may be hysterical or due to St. Vitus's dance, but if each breath on reaching a certain depth is cut short by a sharp pain, there is pleurisy or acute inflammation of the costovertebral articulations.

Entire cessation of respiration for a few seconds is called **apnœa.** People are always apnœic after taking several long breaths, for these breaths have provided a surplus of oxygen, and respiration will not begin again until enough carbon dioxide has reaccumulated in the blood to stimulate the respiratory centre. The best example of apnœa, however, is

seen in **Cheyne-Stokes breathing.** This is due, however, to a decreased sensitivity of the respiratory centre, which can be roused to activity by a marked accumulation of carbon dioxide in the blood. Cheyne-Stokes breathing is characterized by the alternation of periods of apnœa with periods of respiration (Fig. 2), and the respiratory movements, at first quiet and shallow, increase by a definite crescendo in depth, rate, and effort to a real dyspnœa with noisy, sighing, or snoring breaths, after which they become progressively quieter, easier, and shallower until the next period of apnœa begins. The periods of apnœa may last a few seconds or even a minute and the periods of dyspnœa vary much in length, but in any one case or any one occasion the length of these periods is fairly uniform.

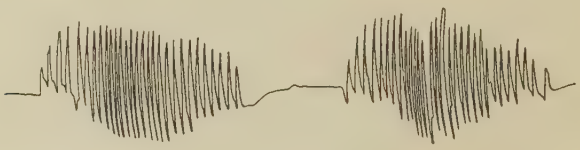


Fig. 2.—Respiratory movements in a case of Cheyne-Stokes breathing.

Cheyne-Stokes respiration is observed in two well marked groups of cases; the first, those with **increased intracerebral pressure,** due to cerebral hæmorrhage, cerebral tumor, etc., all very serious conditions; and the second, **the arteriosclerotic group.** In cases of the latter group the Cheyne-Stokes respiration may continue for months and be compatible with fairly good health. Such patients may be able to eat or talk, etc., only during their periods of apnœa. In a less severe case of this type this form of respiration may be present only while the patient is under the influence of some sedative drug. Its appearance then often startles the nurse in charge of the patient. After the effects of the morphia, etc., have worn off, the respiration will again be regular.

Biot's mode of breathing (Fig. 3) is characterized by periods of apnœa from five to thirty seconds in duration, each followed by a series of rather deep breaths which are

quite irregular in force and rhythm. This mode of breathing is seen especially in meningitis, but it may develop in various conditions which otherwise would show Cheyne-Stokes respiration. Unlike Cheyne-Stokes respiration the successive periods of apnœa and dyspnœa may vary much in length, and there is no crescendo and diminuendo of the respiratory movements.

Stertorous breathing (snoring), produced by the vibration of the relaxed soft palate of a patient with nose and mouth both open, characterizes the sleep of some very healthy persons, though the majority of those who snore are mouth breathers because of hypertrophied adenoid and tonsils. Snoring has diagnostic importance in cases of paralysis of the soft palate and is common in the coma due, e.g., to uræmia, apoplexy, etc.

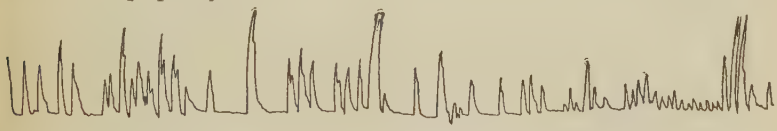


Fig. 3.—Respiratory movements in a case of Biot's breathing.

Stridulous respiration, a medley of harsh, hissing, or whistling sounds, is due to partial closure (stenosis) of the larynx or trachea, caused by disease (e.g., diphtheria), foreign bodies, etc., or by spasm of the larynx. This in children is known as "child crowing" or "spasmodic croup," a nervous condition.

Pulse

Every beat of the normally functioning heart sends a wave along the arteries to the capillaries. This wave is felt as a pulsation of the artery's wall and is called the **pulse.** We count the pulse as the easiest way of getting a general idea of the condition of the circulatory organs. By means of it we can usually determine the heart's rate, its regularity in force and rhythm, approximately the pressure of the blood within the artery, and, finally, the condition of its wall. Usually the pulse is studied at the wrist, on the radial artery. This vessel is convenient, lying, as it does, just under the skin, and

resting directly on a bone, the head of the radius, which serves as a solid foundation for it. As might be expected, this artery may present many anatomical variations. It may be found on the lateral or even the dorsal surface of the head of the radius, or (this may lead to serious error) it may have branched unusually high on the forearm. In such a case one finds in the usual position a small branch and, not suspecting this abnormality, decides that the radial pulse is very small in volume and therefore is weak.

The arteries at both wrists should always be compared since it is not likely that both would be abnormal, and since difference in volume and time of the pulses may indicate serious lesions in the arch of the aorta or along the subclavian or below.

We might, of course, choose for examination any other palpable artery which rests on a firm base, e.g., the temporal, the brachial, the femoral, or the facial artery where it crosses the ramus of the jaw, but one should as far as possible always examine the same vessel, as there is a great deal in "getting used" to one. Only when this is done can one appreciate minor differences.

To count the pulse rate one palpates the artery with the tips of two or, better, three fingers, exerting that degree of pressure which makes the pulsation most distinct. The patient's forearm should, if he is sitting, rest comfortably on a table; if he is in bed, his entire arm should lie flat and limp beside him. All strained, tense, or uncomfortable postures should be avoided. If the patient is excited or apprehensive, the pulse rate will certainly be fast. As a rule one counts for a quarter of a minute and multiplies the result by four. If, however, the pulse is very irregular, it should be counted for a whole minute. One does not count with the thumb, because as that has a good pulse of its own, the observer might unconsciously count his own pulse.

The pulse rate of a normal man is about 72 beats per minute, of the normal woman about 80. The rate depends somewhat on the position of the patient. For instance, the

pulse rate of a normal man is about 66 when he is lying down, 71 when he is sitting, 81 when he is standing, and even more when he is walking. This is the reason why we insist that patients with heart troubles shall lie flat as much as possible. They thus save the heart about 15 beats a minute, 900 an hour, 21,600 a day. The rate depends on the age also. **At birth** the pulse rate of the normal child is from 124 to 144 per minute. It gradually becomes slower until puberty, when the normal rate is from 72 to 92. In purely nervous cases there is a slight tachycardia, from 90 to 120 per minute, which may persist for months, but this rate is greatly accelerated by trivial causes, by exertion, the emotions, etc. In this group belong also the cases of "tobacco heart" and the so-called "athlete's heart" (provided one can rule out hyperthyroidism). The rate is slower during sleep.

The rapidity of the heart is controlled at the base of the brain through the vagus and sympathetic nerves. The latter quicken it. It is the vagus which keeps it as slow as it normally is. Yet the ventricles alone would beat still slower, from 26 to 28 times a minute. **In the Stokes-Adams disease** this is about the rate one finds. In cases of this a little band of muscle fibres, the "wires" over which the contraction passes from the auricle to the ventricle, is so injured by disease that but only a few stimuli can pass through. This condition is called **heart block.** The result is that the auricle continues under control of the brain and beats almost the normal number of times, but the ventricle is, in an extreme case, entirely independent of the auricle, and so beats from only 26 to 28 times a minute. If the heart block is not complete, a few of the auricular stimuli do pass, with the result that the rate of the ventricular pulsation, and hence of the pulse at the wrist, is 28 plus the number of those beats which are due to the stimuli that succeed in passing from the auricle to the ventricle. In heart block we can see the veins in the neck pulsating more than seventy times a minute (since the pulse of the veins comes from the right auricle) while we are counting a very slow pulse at the wrist. Patients with the Stokes-

Adams disease have frequent fainting spells. **Complete heart block** is suggested when the pulse rate is regular but below thirty to the minute, and **partial** when some of the beats are separated by unusually long pauses during which there is no suggestion of a beat.

A multitude of causes can increase the **heart's rate.** When this is very high the condition is called **tachycardia. Among the causes of tachycardia** are the following: hard muscular work; mental influences, as emotions, surprise, fear, delight, etc.; certain drugs, as alcohol, caffein; and fever, of which an increase in the heart rate is as much a symptom as is the rise of temperature. This change of heart rate in **fever** bears a definite relation to the amount of rise of temperature, the ratio being generally 10 beats for each rise of 1° F. of body temperature. One result is that the plotted curve of the pulse rate will, as a rule, run almost parallel to that of the temperature. In a case with fever, no matter how high the temperature, one has little reason to worry about the temperature so long as the pulse is fairly low; but let the heart rate rise to 140 or over, and the outlook, whatever the cause of the fever, becomes at once more serious. Variations from the pulse-temperature ratio may be important in diagnosis. A **pulse rate relatively higher** than the temperature requires may indicate a definite weakness of the heart, pulmonary tuberculosis, even mild cases, sepsis, pneumonia, etc.; in such conditions the pulse may be relatively faster than the above ratio even when the temperature is but slightly elevated or normal. On the other hand, the **pulse rate is relatively too slow** in cases of uncomplicated typhoid fever of adults. In yellow fever the relatively slow pulse has considerable value in diagnosis. In meningitis and other conditions with a considerable increase of the intracerebral pressure the pulse may be relatively or absolutely slow. A rapid pulse with a low temperature is the rule in cases of serious "shock" (collapse) due, for example, to accident, to severe hæmorrhage, to peritonitis, etc.

During a break in cardiac compensation the pulse is nearly

always rather rapid. Of exophthalmic goitre a rapid pulse is one of the cardinal symptoms. A rapid pulse and normal temperature occur in **paroxysmal tachycardia,** a particularly interesting condition natural to some persons. This condition manifests itself by sudden attacks during which the heart suddenly begins to beat fast, exactly twice or exactly three times as fast as before, therefore as a rule about 160 per

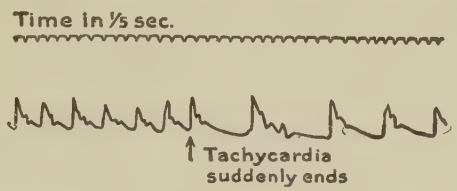


Fig. 4.—Paroxysmal tachycardia.

minute; then after a few minutes, hours, or days of this tachycardia the rate suddenly returns to its former level. These apparently healthy, though nervous, persons usually are painfully aware of the racing of the heart. Somewhat similar attacks are features of some cases of myocarditis (in these cases, however, the patient may be quite unaware of them), but in paroxysmal tachycardia there is no heart dis-

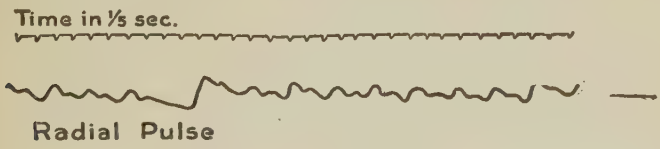


Fig. 5.—Auricular flutter.

ease or disease of any kind as cause. These paroxysms may recur at intervals for years (Fig. 4).

Auricular flutter (auricular tachycardia) (Fig. 5), a by no means normal condition, may be suspected if the pulse rate is persistently 120 or over, and if this rate is not materially modified by rest, movement, or emotion, and if the patient is subject to short attacks of tachycardia accompanied by palpitation or by transient loss of consciousness.

In cases of **bradycardia** (abnormally slow pulse rate) the rate is below 50 per minute. One must be sure, however, that the heart rate really is slow—that the low count is not due to the fact that many feeble beats do not reach the wrist. The commonest cause of bradycardia is jaundice, the bile in the blood being a poison to the heart. It occurs also during the convalescence of fevers, typhoid especially but also many others, and is important even in the early diagnosis of yellow fever. The pulse is chronically slow in myxœdema, and in all conditions which increase the intracerebral pressure, as fracture of the skull and brain hæmorrhage; it is the conspicuous feature of Stokes-Adams disease; it is a warning that a patient has received too much digitalis; it develops in many chronic debilitating diseases, etc. One often hears of "normal" per-

Fig. 6.—Normal pulse wave.

sons (e.g., Napoleon Bonaparte), sometimes of whole families, with very slow pulses, but the heart rate in these cases is practically always above 50 per minute. That is, they have a "slow normal" pulse.

After counting the pulse we notice the **character of the pulse waves.** The normal wave feels much as its diagram would suggest (Fig. 6); that is, it has a fairly rapid rise and a slower descent. The dicrotic wave on the descending limb, which is due to the closure of the aortic valve, is often palpable, provided very light palpation is employed. It is best felt in cases with a low diastolic pressure. It has definite diagnostic value in typhoid fever but may be conspicuous also in cases of severe hæmorrhage and in conditions of peripheral vasomotor dilatation and of cardiac weakness. It should not be confused with a pulsus alternans or with one which has many extra systoles (Fig. 7).

The pulse may be "big" and "bounding" or "small" and "feeble." The size of the pulse wave depends on the size of the artery palpated and on the pulse pressure. A pulse is small or large according as the pulse pressure is little or great, and its significance will depend on the reason for the abnormal pulse pressure, i.e., whether the systolic has risen or the diastolic has fallen.

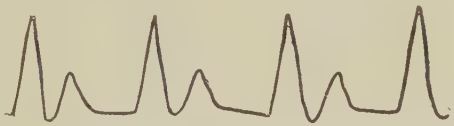


Fig. 7.—Dicrotic pulse.

The **large bounding pulse** (pulsus magnus) (Fig. 8), present in fevers and in cases of cardiac hypertrophy with vigorous heart action, is generally due to a rise of the systolic pressure but may be due to a lowering of the diastolic pressure. This pulse was called "pulsus plenus," a term suggested by the feeling of overfulness of the artery. It is often present in antemortem conditions and was well exemplified in the cases of "blue pneumonia" so common in the in-

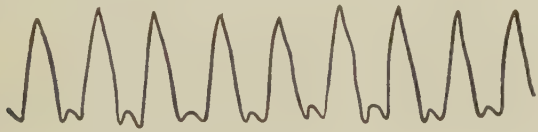


Fig. 8.—Large pulse.

fluenza epidemic of 1918. In these cases the diastolic blood pressure slowly dropped towards zero while the systolic blood pressure was falling much more slowly. In cases of pulsus plenus the pulse waves are very poorly sustained, that is, the blood vessel feels collapsed between beats. The "largest" pulse is that of aortic insufficiency, because in cases of this pulse pressure is the greatest (e.g., S. B. P. 170 and D. B. P. 60 mm. Hg.).

A small pulse (pulsus parvus) (Fig. 9), one which feels

small, threadlike, empty, or running, is found in cases of weakness of the heart muscle due to any of the various functional and organic heart diseases; in cases of extreme circulatory depression; in cases with abnormal rigidity of the arterial wall; and in those with relatively high diastolic and low systolic pressures (seen in conditions which tend to raise both pressures but cannot raise the systolic because of heart weakness). Sometimes the heartbeats are so feeble that the pulse waves can scarcely be felt, or actually cannot be felt, at the wrist. This feebleness occurs near death from any cause; in lesser grades it is continuously present in some cases of mitral heart disease; and it is the rule in Addison's disease. A feeble rapid pulse is a serious sign, but when the pulse is feeble and not rapid one should carefully examine the wrist, for a person with such a pulse may have an unusually small radial artery. The pulse is small also when there is a small or slow ventricular output of blood, as in aortic and mitral stenosis and in certain cases of aneurism which partially blocks the larger arteries.

The pulse is **quick** when the waves strike the palpating fingers with a quick, sharp stroke of surprising force. The best example of a quick pulse is the **Corrigan pulse or water hammer pulse** of aortic insufficiency (Fig. 48). (A water hammer, the student should remember, is a piece of barometer tube sealed at both ends, its lumen a vacuum half filled with water, which is used in the physical laboratory to demonstrate the buffer function of air.) In a well marked case of this condition the jumping of the arteries is conspicuous on inspection, the pulse can be felt even at the finger tips, and the **capillary pulse** can be seen under the finger nail. To appreciate best the quickness of the wave and also to make it more pronounced when one is doubtful as to its presence, one grasps the patient's wrist in his whole hand, his palm against the anterior surface of the patient's wrist, and then raises his arm to a vertical position. A somewhat similar pulse is present in some normal persons after a warm bath, in severe anæmias, etc.

A slow pulse is not one with a slow rate but one whose

waves reach their crest more slowly than normal (Fig. 9). This is most clearly felt in aortic stenosis, a condition in which, because the aortic orifice is so small, the blood must be slowly squeezed into the aorta. Arteriosclerosis, which hinders the prompt rise and fall of the pulse waves, is also an important cause of lesser grades of this type of pulse.

One next notes the **regularity of the pulse.** The beats of a normal pulse are of almost equal force and are separated by intervals of almost equal length; that is, the pulse is "regular

Fig. 9.—Small pulse.

in force and rhythm." In the case of some normal persons, however, we may be startled by noting that the rhythm is quite irregular, groups of beats which follow each other rapidly alternating with groups of beats with much slower rates. If in such a case the period with quick rhythm is coincident with inspiration and that with slow rhythm coincident with expiration, this condition is normal. This "respiratory variation" is conspicuous in normal children, and the few normal adults in whom it is marked are said to have a "juvenile pulse."

Fig. 10.—Pulsus irregularis of auricular fibrillation.

In some cases, as those with disease of the heart, the pulse is **irregular in force and rhythm.** That is, some of the beats are strong, some weaker, and some are scarcely felt at all; some follow one another in rapid succession, while the intervals between others are longer. There is no regularity in these differences; the pulse is irregularly irregular. This is the condition called **auricular fibrillation** (Fig. 10), pulsus irregularis perpetuus, or delirium cordis ("delirium of the heart"). The pulse rate at the wrist in such cases is usually

between 100 and 120 per minute, but this is not necessarily the heart rate, since some of the weaker waves are too feeble to be felt at the wrist. Such a pulse we must count by listening to the heart. The difference between the heart rate and the pulse rate is known as **the pulse deficit**, and, the faster the rate of the fibrillating heart, the greater becomes its irregularity, and so much the greater becomes the pulse deficit. That in such cases the pulse deficit does increase when the rate increases is important in diagnosis, since there are other cases with pulses superficially quite similar but due to many extra systoles or to sinus arrhythmia, and in these cases any quickening of the rate of the pulse tends to eliminate the irregularity.

In some cases **the irregularity of the pulse is regular.** For example, beats may come "in twos," the "bigeminal" pulse,

FIG. 11.—Extra ventricular systoles.

not uncommon in patients to whom too much digitalis has been given. Or, the beats come "in threes," the "trigeminal" pulse.

In many cases it seems as if an occasional beat were dropped. As a rule, however, no beat is missed, but one beat follows so closely after its predecessor that both are felt as one. The next regular beat, the third beat of this series, will feel too strong, since the heart has had an unusual time to rest. The reason for this pause is that the second beat of this series of three is an **extrasystole** which arose in the ventricle wall and merely added itself to the series of three regular stimuli which came down from the auricle. It took the place, however, of the second regular beat, but prevented this stimulus from causing the ventricle to contract. The third beat came on time (Fig. 11). Extrasystoles cause the most

numerous group of cases with irregularity of the pulse. They often occur in persons apparently healthy, who feel each of these extra beats and become very nervous about their condition. They are sure they have heart disease. Some say that the heart "jumps," others that it "stops." Certain of these persons are merely nervous; others owe their irregularity to the use of tobacco, coffee, tea, etc. In many cases it is caused by indigestion. The person may, however, have real heart disease, in some forms of which (the mitral group) extra systoles are common enough.

In cases of **pulsus alternans** the heart beats are very regular in rhythm but not in force, every second beat being very feeble and the others normally strong. This is always a bad sign. In some cases of alternating pulse none of the feeble beats can be felt at the wrist, so, unless we are on our guard,

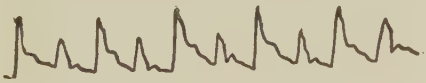


Fig. 12.—Pulsus alternans.

we may count the pulse at half its real rate. Digitalis poisoning may cause this pulse. In such a case the doctor may think the digitalis has "slowed the heart beautifully," whereas really the poisoned heart is beating twice as fast as he thinks. Most of the pulses which are not alternating but feel so, as in typhoid fever, are really pulses with markedly "dicrotic" waves; that is, the feeble wave felt is only a part of one pulse beat. Unless careful, one will therefore count such a pulse as twice as fast as it really is (Fig. 12).

Lastly, by palpation we judge of the **thickness of the vessel's wall.** We first close the artery with one finger and then with the other fingers feel the empty vessel. The radial artery when normal cannot be felt through the skin, but when diseased it becomes thicker and stiffer, so that it may feel, when uniformly thickened, like a rubber tube, or nodular (beaded), a condition due to the presence in the walls of solid, irregular, calcareous plaques. Large plaques may make

it feel "like a goose's neck." Arteries which are sclerotic usually are tortuous also and may appear like a snake under the skin. (The normal temporal artery is always snake-like.) The blood in a thickened vessel may or may not be under high tension.

When the wall of a radial vessel is palpable or any of the peripheral arteries are unusually thickened, an explanation should be found. While it is not true that gradual thickening of the vessels' walls is an almost physiological process, which becomes more and more evident with advancing age, it is true that, the longer a person lives, the more reasons there will probably be for his developing sclerosis. As a matter of fact, a truly normal man of ninety should have as soft arteries as one of thirty years of age.

Vessel walls which are very inelastic easily obliterate the feeble beats, while those which for any reason are partly closed can prevent beats from passing through. The result may be that a man may have "two pulse rates," if the arteries of the two arms are very different.

Blood Pressure

One of the most important of the physical bed-side records is that of the **blood pressure.** Cut an artery, and the blood will spurt six or more feet. This is because the blood is enclosed in overdistended elastic tubes into which it is forced from behind by the heartbeat, while its flow forward is retarded by the resistance of the small vessels. The blood pressure, always highest in the aorta, falls progressively along the arterial tree, as we follow this to smaller and smaller vessels, since the bed of the arterial stream widens as the arteries branch (that is, the sum of the lumens of the two branches of an artery is greater than the lumen of this artery before it branches). We usually measure the blood pressure in the brachial artery just above the elbow.

The systolic blood pressure (S. B. P.) in an artery, that is, the maximal pressure, or the highest point of pressure reached as the pulse wave passes on, is the exact pressure which will just close the vessel so that no blood can pass

through. In the normal man this is about 125 millimeters of mercury (written, 125 mm. Hg. Hydrargyrum is the Latin name for mercury). In other words, if one opens the artery at this point and inserts into it a vertical tube, the blood at the end of each systole will rise in this tube to a point 1700 mm. (or, 5 feet and 7 inches) above the level of the blood vessel (mercury being 13.6 times as heavy as water). **The diastolic pressure** is the level of minimal pressure which the heart maintains in the blood vessels. It is this pressure which each heart beat increases by the pulse wave which it sends down the artery, and the crest of which is the systolic pressure. If the beat should suddenly stop, this pressure would steadily fall to zero as the blood flowed on into the capillaries, but normally each successive beat lifts this falling pressure back to its previous level.

Because of its importance we should be as accurate as possible in determining blood pressure. The instrument used is a **sphygmomanometer** (Fig. 13), consisting of a broad rubber cuff which we fasten around the patient's upper arm, a pump to raise the pressure of the air in this cuff, and a mercury or air manometer to record the exact pressure thus created. After adjusting the cuff about the upper arm we pump air into it until the pulse, felt for at the wrist or listened for with the stethoscope at the bend of the elbow, is just obliterated. We then read this pressure on the manometer. This reading will be the maximum or "systolic" pressure. After we have read the systolic pressure we obtain the minimum or "diastolic" pressure (D. B. P.) by slowly lowering the pressure in the cuff until the sound heard over the artery at the elbow is at its loudest. As a rule this will be just before it suddenly becomes fainter. The difference between the systolic and diastolic pressures is the **pulse pressure. The normal systolic pressure** for men **varies** from 110 to 135 mm. Hg., the diastolic from 70 to 85 mm. Hg. For women the systolic varies from 95 to 125, the **diastolic** from 65 to 70 mm. Hg. **In normal children** the systolic ranges between 90 and 110 for girls (about 5 mm. lower than for boys) and in infants from

75 to 90 mm. Hg. With any intense emotions, e.g., a sudden fear, this pressure rises and may even double itself. We therefore take the reading while the patient is resting quietly, sitting back comfortably in a chair, or preferably lying down, with mind at rest, since even too much curiosity as to what the reading will be will raise it about 20 mm. Hg. The scale should be so located that the patient cannot watch it. Three

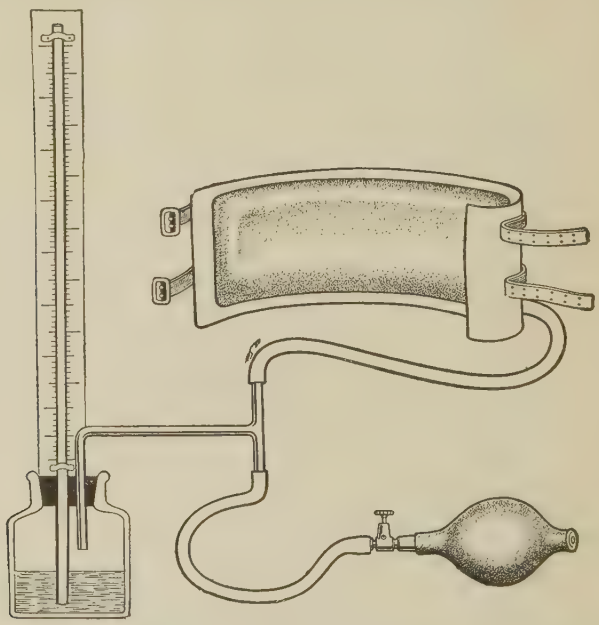


Fig. 13.—The simplest form of a sphygmomanometer, for the determination of arterial blood pressure. This is the Riva-Rocci type, the first of the successful sphygmomanometers, and still perhaps the best.

or four preliminary readings should be made to accustom the patient to the procedure. The lowest of the readings in any one position is the one desired.

It is not "normal" that as we grow older the pressure becomes higher. While it is true in general that a man's pressure is "100 plus his age," this is not natural, and the reason for this is that, the older a person is, the more chances he has had to develop diseases which tend to raise the blood

pressure. Indeed, a man of ninety, if "normal," would have a systolic pressure not over 135 mm. Hg.

In all cases one studies together the systolic, the diastolic, and the pulse pressures. We need all three. As a rule a high systolic pressure and a high diastolic pressure go together, but not always.

A man has **elevated blood pressure** (hypertension) when the systolic is over 135 mm. Hg., a woman if her systolic is over 125 mm. Hg. In the majority of these cases the pressure ranges from 150 to 220 mm. Hg., but in one group it is maintained steadily at 200 to 300, while some have for weeks a pressure of 300 to 320 or even higher. In their early stages these cases of hypertension have a very variable pressure, depending on the frame of mind, degree of fatigue, etc. In one of our cases the systolic pressure while the patient was sitting up was 220. An hour later, after this woman had been put to bed, it was 140 mm. Hg. Later, however, the pressure becomes more "fixed."

Temporary arterial hypertension, a temporary rise of the systolic pressure, often startling in amount, may be due to severe pain, as in gallstone colic, to worry, anger, or some other tense emotional state. Under all such conditions the diastolic pressure may be scarcely affected. **Acute paroxysmal hypertension,** due to excessive vasomotor spasm, is observed in lead colic, and it is greatest (from 250 to 300 mm. Hg.) in those cases in which the lead poisoning affects the brain; in eclampsia, in cases of which the sudden rise of pressure may portend an oncoming convulsion; during the visceral crises of tabes dorsalis.

Chronic arterial hypertension is more common; it is less important in women than in men, occurring especially near the menopause. It is the rule in obese persons. A slight grade is usually a feature of the nervous cases. It is often present where there is arteriosclerosis, and, theoretically, one would expect that increased peripheral resistance would always cause high blood pressure, yet this anatomical condition may be extreme in cases in which the blood pressure is

continuously normal. Some assume therefore that the sclerosis, to cause hypertension, must involve certain arterial systems, either the renal, cerebral, or splanchnic vessels. In chronic Bright's disease the blood pressure is often, not always, high. In such cases, however, it rises when the kidneys begin to fail to perform their function. Increased intracranial pressure, as that due to fracture of the skull, cerebral hæmorrhage, meningitis, etc., raises the pressure considerably. In these conditions the diastolic pressure also is high, which is natural, since the onward flow of blood is definitely impeded. This hypertension follows the hæmorrhage instead of preceding and causing it. Indeed, the common belief that high pressure leads to apoplexy is hardly justified, though both the pressure and the rupture of the cerebral vessel may be due to the same cause and not related as cause and effect. It is a common observation that the great majority of those patients with very high pressure, always over 300 mm. Hg., seldom have apoplexy and usually die of heart failure.

A high systolic pressure is a very helpful early physical sign of toxic goitre. It is the rule in aortic insufficiency. In the former of these two conditions the diastolic pressure remains approximately normal, in the latter lower than normal. One would expect this, since in the former there is no increased resistance to onward blood flow, while in the latter the resistance is decreased; in both the reason for the rise of the systolic pressure is abnormal vigor of the heart beats.

There is another group of persons with chronic arterial hypertension who give no evidence of any of the above conditions or, in fact, of any pathological condition at all, and whose pressures, systolic and diastolic, are the highest seen, the former always over 250 mm. Hg., the latter over 120. This condition, therefore, is called **idiopathic** hypertension or **essential** hypertension.

Chronic vascular hypotension (low blood pressure) is seen oftenest in active tuberculosis (S. B. P. 90 to 100 and D. B. P. 60 to 70 mm. Hg.), of which it is an important physical sign, especially tuberculosis of the adrenal glands (Addison's dis-

ease, in which the S. B. P. is often below 70 and D. B. P. 50 or lower), the glands producing the drug which we use to raise the blood pressure. In conditions causing paralysis of the vasomotor centre (which controls the peripheral vascular resistance) the diastolic may even be 0 while the systolic is normal. Such, during the terrible influenza pandemic of 1918, was the case just before the death of those with blue pneumonia. Sudden temporary drops, usually without danger, are a feature of "faints" and "shocks." To illustrate: Following a prophylactic dose of 1000 units of diphtheria antitoxin the systolic blood pressure of one of our doctors fell promptly to 45 mm. Hg. Two hours later it was 80 (diastolic 62), and four hours later the systolic was 125, the diastolic 75 mm. Hg. This was a good illustration of "proteid shock."

CHAPTER IV

Cells and Tissues

When we aid our eyes by the microscope we find that living tissue is not so homogeneous in structure as we might have supposed but is composed of myriads of tiny units, "cells," which cohere in sheets or in masses. Let us imagine ourselves getting our first glimpse of a brick building from a

Fig. 14.

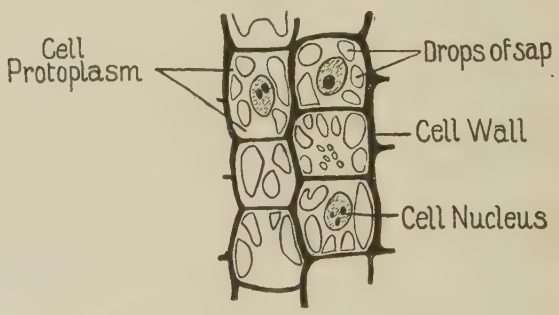


Fig. 15.

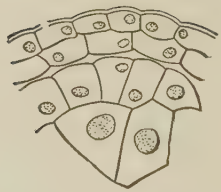
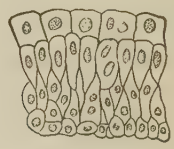


Fig. 16.



Figs. 14, 15 and 16.—Three tissues showing the "brick-wall" arrangement of the cells. (Much magnified.) Fig. 14.—Vegetable cells with thick walls from the cortical layer of the root of the Crown Imperial. (Modified from Hertwig.) One can easily see why the word "cell" was first used. Fig. 15.—The very tip of a rapidly growing plant root. Fig. 16.—Transitional epithelium from the animal body. A cross-section of the membrane lining the bladder. The upper row of cells is part of the exposed surface.

distance. Its walls would seem composed of a uniform red substance, but, as we came nearer, we should see that they were made up of many individual bricks, all similar in size and appearance, and all firmly held together by mortar. Our first impression of vegetable tissue is that it, too, is fairly

homogeneous, but, when we study it "near to," that is, by means of a microscope, we see that it also looks somewhat like a brick wall or a tiled floor, except that the "bricks" are hollow, their conspicuous walls surrounding a cavity filled with a semifluid substance. For this reason the tissue resembles a honeycomb more than a brick wall. The "bricks" of which animal and vegetable tissue is made are the living cells, first described as the "units of structure" by Schwann in 1830.

Each cell (Figs. 14, 15, 16) is a cavity enclosed by a **cell-wall** and filled with a semifluid substance, the **protoplasm**, in which is a solid body, the **nucleus.** Since the first conception of these units was that of a small, closed cavity, the most suitable name was "cell." Later it was seen that not the cell-wall but the semifluid contents, the living protoplasm, was really the important part of the cell. In animal tissue the cell-wall is often very thin or scarcely demonstrable, yet the name "cell" is used here also.

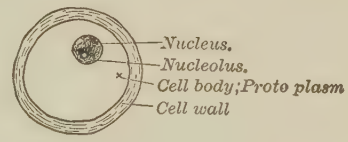


Fig. 17.—A diagram of a single cell.

The animal body consists of **organs**; bones, muscles, a stomach, a liver, a brain, etc., each organ being a structure designed to perform one or more special functions. Every organ consists of vast numbers of cells. Not all cells have the same function, nor do all look alike; but those which do have the same function do look alike, and they are grouped together in masses called **tissues.** For illustration, we have muscle tissue, liver tissue, nerve tissue, etc. Each organ is composed of several tissues, but there is always one which is peculiar to itself or rather, to its kind (there are several organs of the same kind in the body; many muscles, many bones, two eyes, etc.), while the other tissues are present to serve this peculiar one. For illustration, each of the muscles is an organ whose function it is to contract. The real muscle fibres of the muscles are the specialized tissue which has this peculiar property of contractility. A muscle, however,

could not be made up of muscle fibres alone. These fibres must be bound together into bundles by strands of connective tissue; these bundles of muscle fibres are strengthened by elastic tissue, and are bound to the bones, which it is the function of muscles to move by their contractions, by strong bands of fibrous tissues; the muscle fibres must be fed by blood vessels, each composed of several tissues, and must be controlled by nerves, each containing the peculiar nerve tissue. Thus we see that in one muscle there are many tissues which are so arranged that this organ may contract usefully and efficiently.

Tissues, or masses of similar cells, are the building materials of the body. For example, a house is built of several materials: brickwork, which is used in the walls, the chimney, the fireplaces, etc.; iron, which occurs as framework, railing for the balcony, etc.; wood, which is found in floors, window frames, and doors. Yet brickwork, wherever it is found, is always composed of single bricks. However much the parts of the woodwork may differ, it is composed as a whole of many single sticks of wood. The single bricks are the units, the wall of bricks is the tissue. So in the body there are many tissues, and these tissues are composed of myriads of similar single cells. Some tissues occur in but one organ, as the liver tissue, pancreas tissue, brain tissue, etc. Some tissues occur in several organs, as connective tissue, elastic tissue, nervous tissue, etc. Figure 18 represents an artery cut across. An artery is an organ formed to conduct blood flowing under pressure. We see here several tissues, each composed of many cells. The internal surface of the artery is lined by the **intima,** *A*, a single row of endothelial cells. This is a suitable surface for the blood to flow against. Then comes the **media,** *B*, a thick muscle wall upon which the strength of the artery depends, and which also determines the vessel's calibre. When these muscle cells lengthen, the artery's "lumen," its internal circumference, increases; when they shorten, that is, contract, it decreases. Among these muscle cells is a meshwork of elastic-tissue fibres which add much

strength to this muscle wall. The outermost layer is the **adventitia,** consisting of bands of connective tissue, which bind the artery to the structures through which it passes.

All the cells of a tissue—all the liver cells, for example—are practically the same in size and appearance; those of the different tissues, however, liver cells, pancreatic cells, brain cells, etc., differ considerably in appearance.

A cell is, in a sense, an independent structure, of which its nucleus is, so to speak, the "brain"—that on which the cell's life and activity depend. The protoplasm is the cell's body.

The cells of a tissue are arranged in columns or sheets and

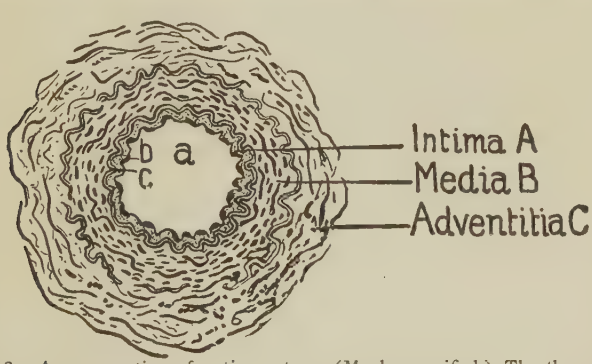


FIG. 18.—A cross-section of a tiny artery. (Much magnified.) The three concentric layers of the wall are, the intima *A*, the media *B*, and the adventitia *C*. The lumen, in which the blood flows, is indicated by *a*, while *b* is the layer of flat cells which lines the lumen, and *c* the strong membrane of elastic tissue between intima and media.

are surrounded on all sides by the tissue lymph, which is as much their atmosphere as the air is the atmosphere for man. From this circumambient fluid, the tissue lymph, the cells get their food and their oxygen. To it they give up their waste products. The tissue lymph, in turn, is in immediate contact with the circulating blood and acts as a go-between for this and the cells, receiving from the blood food and oxygen for the cells and returning to the blood all that the cells have poured into it.

Each cell has one or more functions, usually of a **very special nature,** to perform, and nearly all cells are capable of considerable activity. Some work almost continuously, as

the kidney cells; some intermittently, as the muscle cells and cells of the stomach wall; and some, like the wire mesh of cement walls, or like scaffolding, bind parts of an organ together, as the connective-tissue cells. The work of one kind of cells differs from that of other kinds of cells. The function of the muscle cells is by changing shape to contract; that of the renal cells to separate impurities from the blood; that of the gland cells to manufacture new substances from raw materials supplied by the blood. (Some of the gastric cells, for example, make pepsin). Each cell while in activity is a small furnace which burns fuel and hence produces heat. Each cell requires the products of the work of other cells in order to do its own work. To illustrate: no brain cell could function unless it had at its disposal insulin from the pancreas cells and thyroxin from the cells of the thyroid gland. Each cell lives and dies; each cell is constantly wearing out and needs new material with which to renew its structure; each demands its quota of food and oxygen and will starve or suffocate if deprived of either of these; each must rid itself of its excreta ("ashes") and will die if these accumulate. If one cell of some tissues, not all, dies, a similar cell in its neighborhood may grow, divide into two cells, and thus replace it. Some cells can move, but most cannot. Each cell has its own individual work to do, and its success in doing it depends on its health.

Thus each organ is built of tissues, and each tissue is a colony of tiny individuals. Our body is a confederation of these colonies. The tissues of each organ have fairly separate, definite, functions, the sum of the activity of all their cells, to perform, and they perform these for the whole body. This division of labor is like to that of the people of any large city. In each city there are carpenters, masons, tailors, shoemakers, etc., each man in a trade-union, each with a special work, which alone he can do well, each depending on others for those supplies which, by the division of labor, it is the special work of others to furnish him, and each working for the good of the whole city. So tissues, each composed of

myriads of similar cells, are built up into the organs which we can see and feel, and these organs are built up in turn into an individual man. Some organs are unique and single, as the brain, the liver, the heart, the pancreas, etc.; some are double, as the eyes, the lungs, the kidneys; some are multiple, as the bones of the skeleton, the muscles, the lymph nodes, etc. Some organs are membranes which invest groups of organs, as the skin, the lining membranes of the bronchi, stomach, intestines, etc.; other organs, as the nerves and blood vessels, ramify and penetrate to all parts of the body, binding and coördinating the many different organs together into a unity, a person. In speaking of diseases we usually speak in terms of organs, saying "brain" disease, "stomach" trouble, "liver" disorders, etc., though diseases primarily are disturbances of tissues, that is, of masses of similar individual cells. The diseases of these tissues it is our purpose now to explain.

CHAPTER V

The Blood and Its Diseases

The Normal Blood

The blood is the fluid which is constantly circulating through the body in a system of closed tubes, or blood vessels—the arteries, veins, and capillaries. In total amount the blood makes up about one twentieth of the total body weight; that is, an adult weighting 100 pounds has about five quarts of blood in his blood vessels.

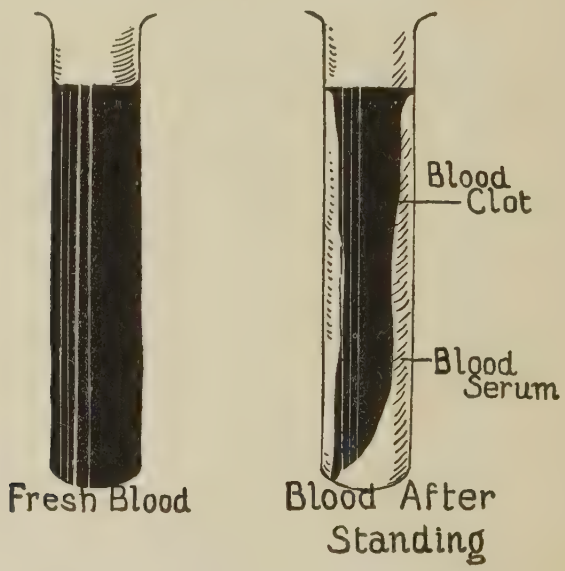


Fig. 19.—Two test-tubes containing blood. The one to the left contains fresh blood. The blood in the one to the right has clotted.

Allow a little fresh blood to stand in a vessel, and soon it is no longer a fluid but a fairly solid, jellylike mass. The blood has clotted; **coagulation** has occurred. Soon this red clot begins to contract, and in so doing it squeezes out drops of a clear yellow fluid, the **blood serum.** The amount of serum increases as the clot shrinks to smaller and smaller dimen-

sions, until the clot is only half as large as at first. Thanks to such coagulation any blood vessel that is cut or ruptured is soon closed up by the clot in the wound, and the loss of blood, the hæmorrhage, is checked. In some persons the blood will not clot, and even a small cut may lead to a fatal hæmorrhage. Normal blood clots whenever it touches a foreign body or is exposed to the air. Before coagulation the blood consists of the **plasma,** a limpid, straw-colored fluid, in which swim the blood cells, or **blood corpuscles**; after coagulation, it consists of the serum and the clot. The latter is composed of the blood corpuscles entangled in the meshes of a network of fibrin, a stringy substance formed in the blood plasma. **Blood plasma,** therefore, is the fluid part of the blood before it clots, and **blood serum** the fluid part after it clots.

By **blood corpuscles** we mean blood cells. They are of three varieties—the red blood cells, which give the red color to the blood, the white cells (leucocytes), and the platelets.

The functions of the blood may be stated briefly as follows: to replenish and to distribute the tissue lymph, the "atmosphere" in which all tissue cells lie; to provide food, raw materials, and oxygen to the cells of the body; to remove the waste material and the carbon dioxide which these same cells have poured into the tissue lymph; and to equalize the heat of the body. In addition, it contains the internal secretions on which much of the health of the body depends, and various protective bodies which prevent blood poisoning. Thus the blood ministers to the health, comfort, and protection of the entire body.

The first function of the plasma we would mention is to **provide the cells with water and food.** As has been stated, each cell requires its quota of food and oxygen, just as does a living person. Deprive it of one of these, and it will die either of starvation or suffocation. It gains its food through the tissue lymph from the blood plasma, its oxygen from the red blood cells. Each cell requires materials of three varieties—protoplasm, raw materials, and fuel. Because of its activity it is constantly wearing out and hence needs new material

with which to repair itself. It can repair its protoplasm only from protoplasm and must have the constituents of protoplasm provided for it, since the animal cell is practically unable to build up this substance which plants build so easily. But the cell requires also raw materials with which to do its work. Some cells are designed to produce heat and hence require fuel to burn. It is the duty of other cells to manufacture for the body special substances called secretions (e.g. the stomach cells produce gastric juice; the liver cells, bile; the thyroid gland, thyroxin; the pancreas, insulin; etc.), and they must have raw materials from which to make these secretions.

The cell, therefore, somewhat resembles a steam engine. An engine, constantly wearing out, requires steel for its repair and it requires fuel also. Or, to change the figure, the cell resembles a mill which uses raw cotton and turns out cloth. The steel becomes a part of the engine, the coal does not; nor does the raw cotton become a part of the mill. Coal and cotton are merely used by the engine and by the mill. So from the blood the cells gain **new material for** their **repair, fuel** to burn in order **to liberate energy,** and **raw material** from which to manufacture **their secretions.**

The plasma also **removes the waste** of the tissues. Each cell is a tiny furnace and by burning fuel produces heat. Just as the ashes of a large engine must be frequently removed, so the waste of the little cell must be carried off, and this the blood stream does. Later the kidney cells remove this waste from the blood.

A third function of the plasma is to **distribute the internal secretions.** By "secretion" is meant the special substance which a cell manufactures. To illustrate: The secretion of the stomach is the gastric juice, a fluid resulting from the action of many cells in the mucous membrane of the stomach. Some of them make the hydrochloric acid, some make the pepsin, some the other constituents of the gastric juice. These various substances—pepsin, acid, etc., are not in the blood but are manufactured by the cells from raw materials

which they get from the blood. They are the secretions of those cells which are in the stomach wall. Gastric juice is an **external secretion** because it is poured into an open cavity of the body, in this case the stomach. Other secretions are poured into the blood stream itself and are known as **internal secretions.** The latter are more difficult to isolate, but their presence is easy to prove, since the body very quickly suffers when deprived of them. For instance, diabetes mellitus is a disease the essence of which is that the cells of the body cannot easily burn glucose, their chief fuel; this unused sugar accumulates in the blood and must be removed through the urine; the cells cannot use sugar unless the internal secretion of the pancreas, insulin, is present. It makes no difference where the pancreas is provided its internal secretion can reach the blood. If the pancreas is successfully transplanted under the skin of the leg, for example, the animal will not become diabetic. The external and internal secretions of an organ are often totally independent. The pancreas produces also an external secretion, pancreatic juice, which is poured into the intestine and is very important in the digestion of food. Another example of an internal secretion is that of the thyroid gland in the throat. Remove the thyroid, and the patient becomes dull, then stupid, and finally an imbecile. His skin becomes thicker and thicker, for beneath it is deposited a considerable amount of mucilaginous fluid, whence the name of the disease produced by hypothyroidism, "myxœdema." But let this patient, even after his mind seems entirely lost, eat every day a little of the thyroid of some animal, and he will recover and will remain well. These internal secretions contain certain very definite and specific (i.e., produced nowhere else) substances called "hormones" (meaning messengers), because their one function is to furnish indispensible aid to organs distant from the one producing them.

The plasma also **distributes the tissue lymph.** The distribution of the water of the body is an important function of the blood, since each cell rests in an atmosphere of this

fluid. When this distribution is disturbed, as in Bright's disease, we may have dropsy.

A fifth function of the plasma is to **distribute heat.** Most cells are little furnaces, burning fuel and hence producing heat, and the constantly high temperature of our bodies is the result. Those parts of the body where most heat is produced are the liver, other large glands, and the muscles. Were it not for the circulation these organs would become very hot while at work and too cool when at rest. The blood becomes heated in these organs and then flows on into other and cooler organs, both those which produce little heat themselves, as the brain, and those which lose much heat, as the external skin. In this way the entire body is kept at a fairly constant and uniform temperature.

In the plasma are also certain **protective bodies.** The circulation ministers to the health and comfort of all these very delicate and sensitive cells, each able to do but one or a few things, each dependent on other cells to supply through the medium of the blood many of its needs. The blood would, therefore, be an ideal home for the many germs—bacteria and other parasites—which so frequently invade it. They are accustomed to live in an adverse environment and to win in a severe struggle for existence. To these hardy individuals an environment such as our helpless body cells enjoy would be a paradise. It is necessary, therefore, for our bodies to have protective substances which will kill these germs without injuring the cells. As a result the great majority of germs of "blood poisoning" are quickly killed in the blood stream, while others do survive there and win the victory over our bodies. While sometimes the success of such germs may be due to the fact that they are more poisonous or more hardy than others, the usual reason is that the body is at fault in that its protection has for some reason weakened. Again, a person who has had typhoid fever, smallpox, scarlet fever, or measles very seldom has the disease a second time. This immunity is due to certain protective bodies, "immunity bodies," some of which may be in the plasma.

The blood performs many other very important functions. We cite the above as instances of its work.

The blood plasma is the fluid of the blood. When the blood is exposed to the air, "strings" of fibrin "crystallize" from it and catch in their meshes the blood corpuscles, thus changing the fluid blood to a solid clot from which later the serum separates. The plasma is by all means the most important constituent of the blood. The red blood cells carry the oxygen and aid in the removal from the body of the carbon dioxide, and the leucocytes are a protection to the body. Both may have functions which we do not yet know or suspect, but important functions of the blood plasma are manifold.

The intestinal wall is the chief plasma-building organ. It is here that it receives its chief constituents, the proteins. We eat in our food a great variety of proteins—fish, meat, fowl, vegetables, etc. The digestive fluids break up these complex substances into much simpler substances, among them many amidoacids. Some of these the intestinal wall builds up again into the proteins of blood: serum albumin, serum globulin, and fibrinogen; others remain in the blood as some of the raw materials of use to the cells when they repair their worn structures.

The same thing happens to sugars. The variety of sugars and starches that we eat is great, but in the stomach and intestines these are practically all changed to one simple sugar, "glucose" (called also, since it is found in grapes, "grape sugar"). This sugar is a constant constituent of our blood and is the one sugar used by the tissues as so much fuel.

The liver also is a plasma-former, or at least a plasma-modifier. All the blood flowing from the intestinal wall, and hence containing all the food absorbed there by the blood vessels, must pass in the portal vein through the liver. This organ removes some of this newly gained food from the blood. It removes most of the glucose, stores it up in the liver cells as glycogen, and keeps it until it is needed. The blood which

circulates through our many organs should have a small but constant amount of glucose—not too much, for that seems poisonous; not too little, for the muscles might run out of fuel when in most need of it. So the liver removes from the blood in the portal vein a large proportion of the sugar which it contains after a meal, stores it up, and then doles it out to the blood in just the right quantities. The normal amount of glucose is about one gramme to one litre of blood, or fifteen grains to one quart. That is, the fuel in our blood stream weighs only a sixth of an ounce, though more is available at any instant for use in the tissue lymph.

But the blood which flows from the intestinal wall to the liver has already passed through many other organs and **has accumulated their ashes.** These ashes of proteid combustion, many of them ammonia compounds, are poisonous. The liver removes them from the blood, transforms them into urea, and gives this nontoxic substance to the blood, from which it is removed by the kidneys. The liver also removes from the blood the hæmoglobin set free from the worn-out red blood cells and makes from it the yellow coloring matter of its external secretion, bile. Many other kinds of ashes are removed by the liver.

After the blood leaves the liver it flows to the heart and thence through the lungs, where it loses its carbon dioxide and some water and takes on oxygen. Hence the lungs too help determine the composition of the plasma. The blood is then pumped through the rest of the body.

Thus each cell selects from the blood that which it wants and returns to it whatever it no longer needs. The selective activity of the cells is wonderful. In the stomach wall, for example, the cells pick out raw materials and make from them the hydrochloric acid, pepsin, etc., of gastric juice. The pancreatic juice, a very different fluid, is made from the same plasma by the cells of the pancreas. The cells of the kidney pick out from the plasma the ashes only and leave all the rest. The muscle cells pick out the fuel necessary for their contraction. The brain cells are very fastidious in their tastes.

So each cell takes out only what it needs from a plasma which is the same for all. But how different are the products of their activity! And how sensitive some of our cells are! If the circulation of the kidney is cut off for forty-five seconds, the kidney cells will not work for nearly an hour, and the poor urine they first excrete is proof of the amount of injury done by this temporary starvation.

The red blood cells, which swim in the plasma, are small coin-shaped bodies, round on their flat surfaces, *a*, and slightly like a figure 8 when seen on edge, *b*. They are really of a yellowish color, and only when seen in large masses do they look red. They are so small that 3300 of them lying on their flat surfaces in a line, *c*, with their edges touching would reach only one inch, and it would take 16,500 piled one upon another like coins, *d*, to make a column an inch high. There are so many of these cells in the blood that a drop about the size of the head of a small pin contains about five millions of them.

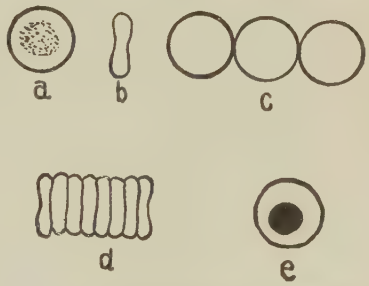


FIG. 20.—Red blood cells. (Magnified 1000 times.) *a*, shows the flat surface of one cell. The shadow in the centre is evidence of its biconcavity; *b*, one cell seen from the edge; *c*, three cells lying flat, their edges touching; *d*, several cells in a "rouleau," or piled like coin; *e*, a "normoblast" or red blood-cell with a nucleus.

The duty of these cells is to carry oxygen. They do this by means of **hæmoglobin,** a very interesting pigment which forms 95 per cent of their composition. Of this hæmoglobin there are about 14 grammes in every 100 c.c. of blood, or about 1½ pounds in the entire body. **Hæmoglobin contains iron,** which enables it to carry oxygen. The iron is small in amount—only about three grammes, or a tenth of an ounce, in the entire body. The hæmoglobin of some of the smaller animals, as the squid, contains copper instead of iron, and as a consequence their blood is blue instead of red. The green pigment of plants, chlorophyll, is similar to hæmoglobin and like it contains iron.

The framework of each red blood cell is called its **stroma**. This is small in actual quantity, for most of the cell is hæmoglobin, and yet to it the cell owes its solidity, size, shape, and elasticity. If all the hæmoglobin is washed out, the cell is said to be "laked," but the stroma may remain as a pale shadow preserving the cell's size and shape. Many poisons will lake the blood, as snake poison and the poison of many germs; the blood of one animal may be poisonous to another. The serum of a dog, for example, is so poisonous to a rabbit as to kill him. This is the reason why the transfusion of blood from a healthy animal to a sick man is impossible. We do **transfuse our patients** but only from man to man, and we always make sure in advance that the blood of the "donor" is "compatible" with that of the patient. That is to say, each person belongs to one of four (or six) **blood types,** or blood groups, and we must be careful that the donor's blood is of the same type or group as that of the patient. One's relatives may have blood of a quite different type from one's own blood. But **typing the blood,** that is, finding a donor whose blood is of the right type, is not quite enough, since there are minor degrees of compatibility even among those bloods belonging to the same group. When a transfusion is planned, therefore, we call in several prospective donors whose blood is of the same group as that of the patient, and then we test directly the blood of each by mixing a few drops of it with that of our patient, see what the results are, and select that donor whose blood is most compatible with that of our patient. If the two bloods are very compatible we may introduce even six hundred or more cubic centimeters of the donor's blood into the vein of the patient and the latter should at once feel better.

Hæmoglobin has such an affinity for oxygen that when the two are brought together they unite and form oxyhæmoglobin, which is very red. But this combination is very weak, and oxyhæmoglobin readily gives up its oxygen if the tissue lymph has less oxygen than itself. Hæmoglobin has a bluish-red color, whence the difference in color between arterial and

venous bloods. **This affinity of hæmoglobin for oxygen** is the secret of respiration. In the lungs the hæmoglobin of the red blood cells is exposed in a thin layer to the air, which contains about 20 per cent of oxygen. There oxygen and hæmoglobin unite and form oxyhæmoglobin. The blood then circulates back to the heart and thence around the tissues. The tissue cells are meanwhile using up the oxygen which they had and all they can get from the lymph around them. Hence this lymph is poor in oxygen and so has a stronger affinity for it than has the oxyhæmogloblin in the blood vessels. When the oxyhæmoglobin reaches this lymph, therefore, it at once splits, its oxygen going to the tissues, and the hæmoglobin circulating back through the veins to the lungs for more oxygen. Meanwhile the carbon dioxide which the cells give off is carried by the plasma to the lungs. There is very little carbon dioxide in the air of the lungs therefore this gas leaves the blood and takes the place in the air of the oxygen which the hæmoglobin is absorbing. That is why the air we exhale is "bad." This interchange, within the blood tissues, of oxygen for carbon dioxide is known as **internal respiration**; the interchange in the lungs is known as **external respiration.**

The red blood cells are formed in bone marrow, in the case of adults chiefly in the marrow of the ribs. As any cook knows, there are two kinds of bones—those in which the marrow is red and nutritious, "marrow bones," and those containing only fat, which is of no value as food. It is in the red marrow that these red blood cells are formed. They have no definite cell membrane, but when young they do, like all perfect cells, have a nucleus. This nucleus, however, is not necessary in respiration and would probably make the cell too heavy. It is, therefore, by a wise provision, discarded as soon as the cell is mature enough to be used in the circulation. The red blood cells of reptiles, birds, and some animals—camels, for example, all have nuclei. The red blood cells of man, which have lost their nuclei, have lost their most important element, as far as cell life goes. That

is, they have sacrificed their life for the sake of their function. Sometimes when the blood is very much in need of new cells, as after a severe hæmorrhage, the young nucleated red cells are allowed to enter the circulation, but this is not normal, for usually they remain in the marrow until they have lost their nuclei. Red cells remain in the circulation perhaps about six weeks. Interestingly enough, we find in normal blood no old or half-worn-out cells, for before the first sign of age appears the cells are removed and destroyed, perhaps in the spleen, the bone marrow, and the liver. The liver then carefully splits off the iron from the hæmoglobin and saves this for the manufacture of new red cells, but the rest of the hæmoglobin is "thrown away" and appears later as the pigment which gives the color to bile.

We have seen that the one function of these red blood cells is to carry oxygen, a very necessary but still rather simple function, while the functions of the plasma are much more numerous and complicated. In studying the blood in anæmia, however, we judge its condition chiefly by the number and condition of its red blood cells, since they are much easier to study than is the plasma and, fortunately, seem to be a very sensitive index of blood conditions.

The leucocytes also are blood cells, but they contain no hæmoglobin and hence are colorless. They are "perfect" cells; that is, they have a nucleus; they are alive, and able to live a rather independent existence; they move about of their own volition, and rapidly, too. Normally there is about one leucocyte to every 700 of the red cells; that is, there are about 7,000 of them in each cubic millimeter of blood. They are not flat cells, like the reds, but are usually spherical, or fairly so, and they can change their shape rapidly.

The majority of leucocytes—that is, from 70 to 72 per cent of them—**the finely granular cells** are about one third larger than red blood cells (Fig. 21). Their nucleus may be of almost any shape except round, and their protoplasm is filled with very fine, dust-like granules. These cells move by projecting their protoplasm at one point, *a*, and then flow-

ing into this projection, which is called a "pseudopod." They live on that which they absorb from the surrounding plasma, but, when they come across any solid body that attracts them, a germ for example, they "swallow" it by enclosing it in their protoplasm. The protoplasm in contact with the germ then secretes a digesting fluid, which forms around it a clear area in which one can watch the germ fall to pieces and disappear

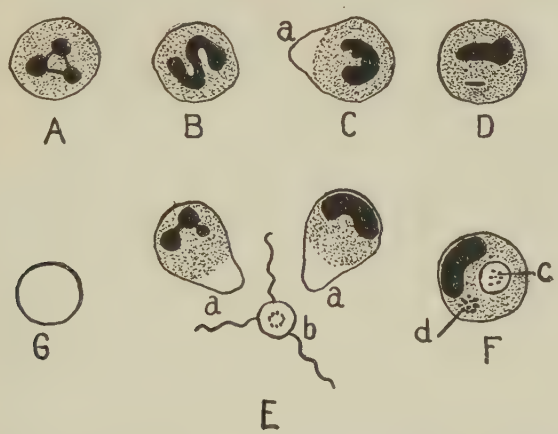


FIG. 21.—Finely granular leucocytes. (Magnified 1000 times.) *A, B,* two leucocytes at rest. *C,* a leucocyte moving. Note its "pseudopod," *a,* of clear protoplasm and that the inert granules and nucleus "collect in the rear." *D,* a leucocyte which has "swallowed" a bacillus which it is now digesting. The clear halo around the bacillus represents the digestive fluid which the protoplasm has secreted. *E,* represents two leucocytes both racing for the same flagellated malarial parasite, *b; a* and *a* are the pseudopods of the leucocytes. *F,* a leucocyte which has swallowed two malarial parasites, *c* and *d,* and is now digesting them. The malarial parasite *c,* is possibly still alive, but only a few pigment granules are left of *d. G* is a red blood-cell introduced in the picture for comparison of size. Note the variety of shapes of the nuclei of the seven leucocytes.

as it is digested. This process of devouring a smaller cell or a germ is called "phagocytosis." The leucocytes seem able to secrete several digestive fluids, which differ according to the tasks they have to perform.

These cells are also the scavengers of the body. If any dust or dirt (Fig. 21) gets into the blood, it is their duty to pick it up and carry it to the ash heaps of the body, the spleen and bone marrow. The malaria germ, for instance, digests the hæmoglobin of the red cells in which it lives and in so doing forms masses of fine dust-like refuse, which later

FIG. 22 *A*.

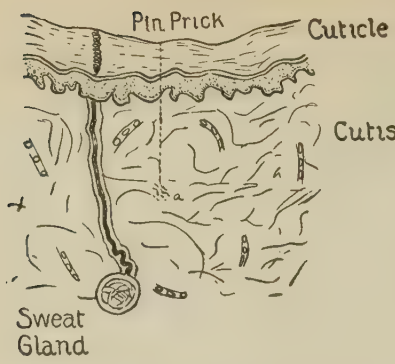


FIG. 22 *B*.

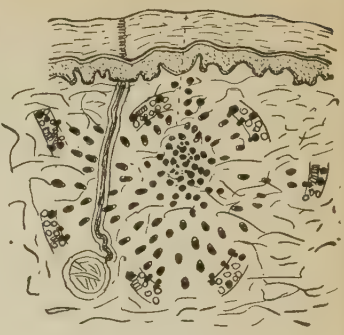


FIG. 22, *A*.—"The History of a Boil." This figure represents a cut-section of normal skin. Note the surface layer, or cuticle, and the "true skin," or cutis. In the cutis one sees that the blood capillaries are just wide enough for the blood cells to pass through "in single file." The skin has just been pricked by a dirty pin. On the point of this pin were several poisonous germs which were deposited at *a*.

FIG. 22, *B*.—"The History of a Boil" (continued). The poison from these germs (*A*, *a*) diffuses through the cutis. The capillaries dilate. The leucocytes force their way through the walls of the capillaries and travel towards these germs. Note the dumb-bell shape of the leucocytes as they pass through the minute holes in the capillary walls, and their pseudopods as they travel towards their common destination, attracted by the poison from the germs. The skin in this region is now swollen, red, hot, and painful.

FIG. 22 *C*.

FIG. 22 *D*.

FIG. 22, *C*.—"The History of a Boil" (continued). The migration of leucocytes has continued until now they form a dense mass surrounding the germs. The poison of the germs has killed all the leucocytes and also all the cutis immediately around them, and now digestive fluids from the dead leucocytes are turning the whole dead mass into liquid pus. The boil has "come to a head." There is a little lump on the skin and through its thin covering of cuticle can be seen the yellow pus.

FIG. 22, *D*.—"The History of a Boil" (concluded). The boil has finally ruptured. The liquid pus has escaped carrying with it the germs and most of their poisons; the migration of leucocytes has stopped; the capillaries are returning to normal size and now new tissue will grow and fill up this hole.

become free. The leucocytes pick these up and carry them to the spleen, where they can be found years later. The leucocytes are also the policemen of the body. When a germ enters the blood they will try to engulf and digest it. Many of them will probably die in the attempt, if the germ produces a very strong poison. To ingest those germs which have gained access to the tissues leucocytes leave the capillaries and travel towards them through the tissues, where they are called "pus cells." Supposing one pricks his finger with a pin which has virulent germs on it (Fig. 22). These germs, *a*, are deposited under the skin, where they liberate poisons which kill many of the tissue cells in the neighborhood and become diffused through the tissue. The capillaries in the neighborhood at once become distended, and the leucocytes of the blood collect in them, make their way through their walls, and move from all sides towards the infected area. There they form a cloud around these germs and seem to try to kill them by devouring them or by setting free germicidal (germ-killing) substances in their neighborhood. At the same time the dead bodies of those of the pus cells which are killed by the germs give off a digesting ferment which liquefies the dead tissue. The finger at first was uniformly swollen, hot, red, and painful; now this collection of leucocytes makes a projection at one point. The surface at this point soon becomes yellow, because the skin there is dead, and because there is under it a large mass of these pus cells. When the digestive process has reached the skin, we speak of the boil as "coming to a head," meaning that the ferment has digested the tissue from the spot where the bacteria are and through to the skin. This soon ruptures, and allows the pus, consisting of all this liquefied tissue, the bacteria, their poisons, and the great masses of pus cells, to escape. Then, if all the germs are gone, the hole is soon filled in by new tissues, and the boil is healed.

The process of engulfing a germ, **phagocytosis,** is best studied in malaria (Fig. 23, *E*). One finds under the microscope an active malarial germ and watches it. An active

leucocyte (sometimes three or four) in the neighborhood will very soon be seen to move towards it, to ingest it, and then to digest it. This attraction, called **positive chemotaxis,** is undoubtedly due to a poison from the germ which is diffused in all directions, and which, when it reaches the leucocyte, attracts it. **Negative chemotaxis** occurs when the poison is so strong or of such a nature that the leucocyte moves as fast as it can in the opposite direction, as if to escape. Phagocytosis, while a very interesting phenomenon because we can see it so plainly, has, nevertheless, probably been much overestimated as a protection of our tissues against disease.

The leucocytes not only fight for the protection of the body but also transport fat in tiny globules around the body.

The leucocytes described above are filled with very fine granules and hence are known as "finely granular cells." Since their nuclei are never spherical but of various other shapes—those of strings fantastically twisted, rods, balls, etc., they are also called polymorphonuclears, whence their real name, polymorphonuclear-finely-granulars, or, since their granules are stained by neutral dyes, **polymorphonuclear neutrophiles.** There is another group of leucocytes (Fig. 23, *A*), similar in size and in nuclei but different in the size of the granules, which are very coarse. These, therefore, are called the polymorphonuclear-coarsely-granular cells, or, since their granules are stained by a dye named eosin, **polymorphonuclear eosinophiles.** Normally these cells make up only two to four per cent of the entire number of leucocytes. They can move more rapidly than do the finely granular cells, and they seem to be an advance guard, for when trouble arises they are usually the first on the field.

In normal blood from 20 to 25 per cent of the leucocytes are the small **mononuclears** (Fig. 23, *B*), which are about the size of red blood cells. They have a relatively large, usually spherical, nucleus and a very narrow rim of protoplasm containing no granules. They are called also **lymphocytes,** since they were once supposed to come from lymph nodes, though probably they all originate in the bone mar-

row. They are sometimes motile, and they seem especially attracted by the toxin of the germ of tuberculosis.

About four per cent of the leucocytes are the **large mononuclears,** or **endothelial** cells (since apparently they come from the walls of the capillaries), which are five or six times as large as a red blood cell (Fig. 23, *C*, *F*, *G*). They have large nuclei, which in shape are oval or indented, and considerable clear protoplasm.

It will be noted that no granular cells with round nuclei are found in normal blood, and yet all the granular cells,

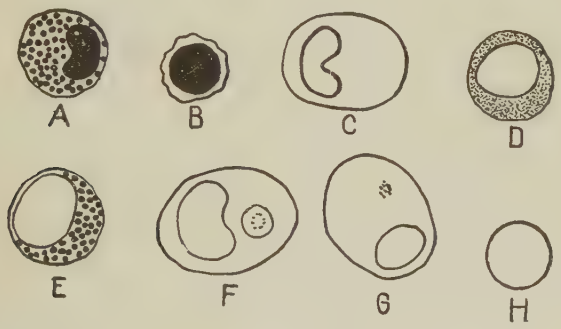


Fig. 23.—Coarsely granular and nongranular leucocytes, and myelocytes. (Magnified 1000 times.) *A*, a coarsely granular leucocyte, or "eosinophile." *B*, a small mononuclear leucocyte, or "lymphocyte." *C*, a large mononuclear leucocyte. *D*, a finely granular mononuclear leucocyte, or "finely granular myelocyte." *E*, a coarsely granular mononuclear leucocyte or "coarsely granular myelocyte." *D* and *E* are never found in the blood of a normal person. They occur normally in the bone marrow. *F* and *G* are leucocytes similar to *C* which have swallowed malarial parasites. *H*, a red blood cell.

when they are young, do have round nuclei, but, like the nucleated red cells, they stay in the bone marrow, where they are formed, until they are mature enough to leave it, when their nuclei are irregular in shape. While their nuclei are still round, these cells are called **myelocytes;** and of course there are "coarsely granular myelocytes" (Fig. 23, *E*) and "finely granular myelocytes" (Fig. 23, *D*). In one disease, leukæmia, both of these forms of myelocytes occur in the circulating blood in considerable numbers.

Normally, there are in the blood from 5000 to 10,000 leucocytes per cubic millimeter. When the count is above

10,000 the condition is called a **leucocytosis**; when below 5000, a **leucopenia.** Not only the total count but also the relative count of these various varieties of leucocytes is an important aid in diagnosis, for with the same total number their percentages may be quite different. Inflammations anywhere in the body and diseases with pus formation often cause a rise in the count, due to a great increase in the polymorphonuclear-finely-granulars. In pneumonia, for example, the leucocytes may be even 30,000 or 100,000 or more per cmm. This increase of leucocytes means, we believe, that the person is making a good, vigorous, fight against the germ of pneumonia. We do not know exactly how the leucocytes fight this infection—whether they devour the germs or not; and no idea of the outcome can be gained from this count, for even after the most vigorous fight the patient may lose, that is, may die. If, however, the patient is weak and the leucocyte count does not rise at all, we believe he is not strong enough to fight in this manner against this disease, and we know that the outlook for him is very serious. In other diseases as surely caused by germs—typhoid fever and tuberculosis, for example, the leucocytes, instead of increasing, decrease in total number. This difference between diseases we cannot yet explain. It is fortunate that there is this difference, for it is a very great aid in diagnosis.

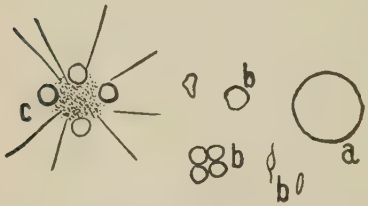


FIG. 24.—Blood-platelets (magnified 1000 times). *a*, a red blood-cell introduced for purposes of comparison. *b*, blood-platelets seen from the side and edge. *c*, a mass of platelets. The most have broken down to a granular mass and from this as a centre strands of fibrin radiate.

In other diseases the polymorphonuclear-coarsely-granular cells are those that are increased. This **eosinophilia** is of great help in the diagnosis of infections by animal parasites, especially "trichiniasis," which we get from raw pork. These cells, however, are increased by any animal parasite, even the common round worms in the intestine. They are increased also in asthma.

In malaria the large **mononuclear leucocytes are increased,** and these, being phagocytic, may contain pigment granules (Fig. 23, G) or whole malarial parasites (Fig. 23, F) in their protoplasm. In other conditions, especially tuberculosis, the **small mononuclears are increased.** By studying the leucocytes, therefore, we receive valuable assistance in diagnosis.

The blood platelets, the third corpuscle (Fig. 24) of the blood, are small colorless cells furnished by some of the giant cells of the bone marrow. They are so fragile that they are seldom seen in fresh blood, but must be demonstrated by special methods. These platelets are certainly **important in blood coagulation.** They are present in the blood to the number of about 250,000 per cmm.

Since the **composition of the blood as a whole** depends on the activity of so many organs, one would expect that diseases of these various organs would greatly influence the blood. This may sometimes be the case, but, strange to say, the blood, so far as we can judge, usually remains remarkably normal, even when there is serious disease. For example, a person with diabetes mellitus, instead of voiding about one quart of urine a day, may void over 25 quarts; instead of voiding with this water about one quarter of a pound of solid matter, the kidneys may void daily as much as two pounds of solid matter, most of which is sugar. Now, all of that water and all of that sugar appearing in the urine have come from the blood, and yet, if during the days when so much water and sugar is being eliminated the blood should be carefully studied, the changes found in it would seem slight indeed.

Diseases of the Blood

Anæmia theoretically means a lack of blood; it really means a poor condition of the blood. Patients who are anæmic look pale—not necessarily their cheeks but their tongue and lips. The color of the cheek, unlike the color of the mucous membranes, depends more on the size of its skin vessels and on the amount of blood flowing through them,

than on the blood's richness. The cheeks of even an anæmic person can blush.

The volume of blood in the blood vessels varies little, for, if a large amount of blood is suddenly lost, as by a severe hæmorrhage, the tissue lymph in which the cells lie begins at once to flow into the vessels and soon makes up for the lost volume of plasma fluid. By anæmia we do not mean a decrease in the amount of blood in the body, though in pernicious anæmia that may be the case; in other forms of anæmia there seems to be an actual increase in the volume of the circulating blood. What we mean is that its quality is poor. Some day, perhaps, the quality of blood will be judged by the amount there is in the patient's body, or, perhaps, by the quality of its plasma; but at present we judge it, first, by the **number of red blood cells actually present in each cubic millimeter of blood**; and, secondly, by the amount of hæmoglobin in each hundred cubic centimeters of blood. The number of red cells is determined by actually counting them. A carefully measured small amount of blood is accurately diluted two hundred times, then spread on a ruled surface one millimeter square and one tenth of a millimeter deep. This ruled square is subdivided by lines into sixteen equal squares. We count the cells in some of these and then calculate the number in a cubic millimeter of undiluted blood. **The hæmoglobin is determined** by diluting (in order that the color may not be too deep) a measured amount of blood in a measured amount of water and then matching the dilute blood against a standard color scale. **A normal man has about 5,000,000 red corpuscles** in one cubic millimeter of blood, a normal woman about 4,500,000. In some anæmias, the count is as low as 200,000, or even lower; in certain other conditions it is as high as 10,000,000. The latter condition is known as **polycythæmia.**

Hæmoglobin is expressed in terms of percentage. A normal person has about 14½ gm. of it per hundred cubic centimeters of blood and, therefore, this amount is called 100 per cent. When we say, for instance, that a person's hæmoglo-

bin is 50 per cent, we mean that each hundred cubic centimeters of his blood contains one half the proper amount of hæmoglobin. When the marrow must build cells with unusual rapidity, as after a hæmorrhage, it produces cells which are smaller and thinner than the normal, that is, cells each of which contains less hæmoglobin than normal. The red cell count in such a case may be almost normal and yet the hæmoglobin percentage very low. The relation between these two values, the percentage of hæmoglobin divided by the percentage of the count, is called the **color index.** For example, suppose a person's count is 4,000,000 cells, that is, four fifths (80 per cent) of the normal number, and the hæmoglobin is 60 per cent. Then 0.75, the result of dividing 60 by 80, is the color index. In certain other conditions the red cells are unusually large and thick. For example, the count might be 1,000,000 cells (20 per cent of the normal number) and the percentage of hæmoglobin 30. The color index in this case would be 30 divided by 20, or 1.5. The color index is important in diagnosis.

By secondary anæmia is meant an impoverished condition of the blood due to some cause which we know, as hæmorrhage, the poison of a fever, etc. In practically all secondary anæmias the hæmoglobin will be found to be more reduced than is the count, and hence the color index is low. We might, for example, find a count of 4,000,000 cells and hæmoglobin of 65 per cent, with a color index, therefore, of 0.7. That is, the count may be almost anything below normal, but the hæmoglobin will usually be still lower. If we examine the red cells under the microscope we see that the reason for this is that these **cells are in general smaller and thinner** than normal. Of course none of the previously normal cells can have shrunken, nor can any have lost some of their hæmoglobin. The truth is that the bone marrow can multiply these cells much faster than it can manufacture new hæmoglobin for them, so that, when there is need for haste in the formation of new cells, as after a hæmorrhage, the marrow divides the hæmoglobin at hand into many light-

weight cells rather than into a smaller number of cells of standard size and thickness. Then too, while the blood is being hastily restored, the overactive bone marrow often allows nucleated reds to enter the circulation. The bone marrow does not work steadily but by "spurts," so that for a while we see no nucleated red cells, then on certain days we see vast numbers of these cells, and this "crisis," as the period of increased production is called, is followed by a jump upwards in the red amount. Since the bone marrow produces leucocytes also, it may, while very actively producing reds, produce too large a number of leucocytes as well. Hence the white count is often over 10,000.

Secondary anæmias are due to hæmorrhage, to certain poisons, especially those produced in acute fevers, to chronic diseases, to poor food, etc. The anæmia due to hæmorrhage may be acute or chronic.

By an **acute anæmia due to hæmorrhage** we mean an anæmia due to one sudden loss of blood. A person can lose from one half to two thirds of his blood, that is, over two quarts, before his life is in jeopardy. Such hæmorrhage occurs when an artery is opened with a sharp instrument, or when a very severe nose bleed occurs, or when a small vessel, e.g. on the floor of a gastric ulcer or in the wall of a tuberculous cavity of the lungs, ruptures. The blood at once restores its volume of plasma from the tissue lymph, and then the bone marrow very actively supplies new cells. To do this takes from one to thirty days, according to the amount of blood lost.

A chronic post hæmorrhagic anæmia is one produced by a series of hæmorrhages occurring at intervals so close together that there is not time entirely to restore the loss due to one before the next occurs. Such an anæmia results from repeated hæmorrhages from the lungs, from ulcerated cancers, from hæmorrhoids, etc. The amount lost each day may be small; the patient may not even know that he is bleeding. It takes a much longer time than in acute anæmia, sometimes eight or ten months after bleeding stops, to recover from such

a condition. Evidently the bone marrow itself has suffered from the prolonged anæmia.

Toxic anæmias are due to blood poisons, either chemicals or bacterial toxins. While they may seem to injure the blood directly, causing a "hæmolytic" anæmia, yet the chances are that for the most part their effect on the blood is the result of injury by them of the organs which produce it. A good illustration is the blood poisoning (septicæmia) which follows an infected cut or an attack of tonsillitis. A few germs, e.g. streptococci, get into the system, multiply in great numbers, and with their poisons rapidly lower the red blood cell count. In these cases there is some jaundice, which does suggest blood destruction. Another example is the anæmia of those who work with white lead or arsenic. Benzol, a poison which is used in some factories, causes an extreme anæmia with very low leucocyte count. Evidently the bone marrow, because of the poison, is quite unable to do its work properly, and hence the name **aplastic anæmia,** which indicates that its cause is lessened production. These patients are never jaundiced. Pneumonia, diphtheria, typhoid fever, acute articular rheumatism, etc., and three important chronic diseases—cancer, tuberculosis, and lues, are important causes of anæmia. Certain intestinal parasites, as the hook worm, may produce a severe anæmia by their poisons as well as by the minute hæmorrhages they cause.

Secondary anæmia is often seen among the poor and is sometimes called the **anæmia of the poor,** a name which suggests that it is due to the poor food and bad living conditions which poverty necessitates. However this may have been in the past or in other countries, we see no evidence of such cause and effect here, and we find that exactly the same kind of anæmia is common among the rich also. In both classes its cause is some latent disease, which of course would be more common and less carefully and efficiently treated among the poor than among the well-to-do. Such patients of the poorer classes may have latent tuberculosis, heart and kidney troubles, nose and throat infections, decayed teeth, and other

ailments, of some of which they may be unaware, and others of which they neglect. Their food is good enough but is badly chosen, poorly cooked, eaten rapidly, and, as the patient works immediately after meals, it is digested with difficulty. The frying pan is therefore an important cause of anæmia. On the other hand, the alimentary canal, because of long standing trouble in stomach, gall-bladder, or appendix, may be unable to digest the food properly. Besides, the state of mind of the patient is important, for a person always worrying cannot digest his food properly. But, after all, the stomach and the blood will stand a great deal of abuse, and we find that infections are usually the cause of this anæmia. Certain medicines cause very severe anæmia, especially the "headache powders" advertised as "harmless" but loaded with powerful drugs.

The above are a few of the causes of secondary anæmia. By far the most important cause for the young is infection, for the elderly, cancer.

By primary anæmia we formerly meant merely an anæmia whose cause was not yet known. Now, however, the term is used oftener of the blood picture, as composed of the blood count, the hæmoglobin estimate, and the appearance under the microscope of red blood cells. The primary anæmias are chlorosis, pernicious anæmia, leukæmia, and Hodgkin's disease.

Chlorosis is a disease of girls from about twelve to fourteen years old. It does occur in older women, especially in those who had it when young. It has occurred in men, but so rarely that the diagnosis is usually open to doubt. The patient may be recognized at first glance by the very pale lips, the greenish color of the skin (hence the name "chlorosis") and the bluish tint of the whites of the eyes. The patients are generally well nourished, and, except for their color, look healthy. They complain of shortness of breath, palpitation of the heart, especially on exertion, and usually of indigestion. The blood shows an anæmia resembling the secondary type; that is, the hæmoglobin is more reduced than

is the number of the red cells. The count is often almost normal but as a rule is 4,000,000 or just above. The hæmoglobin is often between 40 per cent and 50 per cent, and hence the color index is about 0.5. Under the microscope one sees the reason for this low index. The cells are practically all smaller than normal and so much thinner that they look as if they had a hole in the centre, like a doughnut. The leucocyte count is about normal.

The cause of this anæmia is doubtful, but the disease seems to be wholly due to poor blood formation ("aplastic" anæmia) and not at all to blood destruction ("hæmolytic" anæmia). As it occurs at an age when the red marrow of the long bones is becoming fatty, which means a decrease in the amount of blood-building tissue, and also at a time when because of menstruation and more rapid development of puberty there is need of increased blood formation, it would seem as if the tissue left were overtaxed.

The treatment is simple. Patients with chlorosis should stay in the fresh air more than they usually do, should eat more nourishing food, and should avoid the constipation usually present; but especially do they need iron as medicine. The market is flooded with expensive iron preparations —organic compounds, etc., but, while they are all alike in being costly, they are no better than the very cheap Blaud's pills.

Primary pernicious anæmia, as the name implies, is an anæmia the cause of which is not known, and which in almost every case sooner or later ends fatally. It occurs particularly in adults, and since it causes very little loss of weight, such patients often look robust. Their lips and tongues are very pale, their faces are of a brownish or yellowish tint resembling sunburn, and they pass as very healthy-looking persons. They complain of indigestion, shortness of breath, palpitation of the heart, extreme weakness, sometimes a slight swelling of the feet, or a tingling in the feet and hands. It is interesting to observe how many different diagnoses of such cases are made. The patients

come to the clinic with the diagnosis of heart disease, jaundice, Bright's disease, paralysis, or peripheral neuritis, and they usually have been treated for one or more of these diseases. The blood shows an extreme anæmia. The count is often as low as 1,000,000 or even 200,000, but the hæmoglobin is relatively high for such a count, perhaps 30 per cent, and hence the index is almost always over 1 and may be even 1.75, a very important point in the diagnosis. As the index would suggest, the red blood cells are, on the average, much larger and thicker than normal, and yet all sizes are seen and, what is more unusual, all shapes as well. Often one sees **nucleated reds,** especially some, called **megaloblasts** which are larger than any seen in secondary anæmia, and which are very important in the diagnosis. The leucocyte count is low, and there is an increase in the small mononuclears. Because of the jaundice and the deep color of the urine, both due to a great increase in pigments derived from hæmoglobin, this anæmia has been explained as being caused by increased blood destruction and has therefore been called a "hæmolytic" anæmia. Hæmolysis, however, is doubtful. In this anæmia there probably is also a considerable reduction in the total volume of blood in the body.

The cause of primary pernicious anæmia is still unknown, but it certainly is some poison which injures the bone marrow, and there is much evidence that the source of this poison is in the gastrointestinal canal. Patients with primary pernicious anæmia usually have badly infected mouths (pyorrhœa of the gums and chronic infection of the tongue which makes it smooth and glistening); atrophy of the mucous membrane of the stomach, which leads to early absence of the free hydrochloric acid of the gastric juice; and some disease or infection of the intestines, e.g. by animal parasites.

The treatment of pernicious anæmia includes fresh air, sunlight, quiet but cheerful surroundings, freedom from care or worry, rest in bed, and plenty of sleep. All physical and mental fatigue should be avoided. Anæmic patients feel the cold and so should be well protected. The care of the mouth

is especially important since the chronic infections there in part at least explain the disease. The diet is extremely important, and every effort should be made to improve the appetite and to build up the strength by nutritious food. Since in this anæmia periods of good digestion alternate with those of bad, advantage should be taken of the former. The diet should be plain, easily digested, and rich in iron, such as eggs, spinach, fish, meats, etc. Since achylia gastrica is the rule of pernicious anæmia, acid fruits may have considerable value. Free purgation is, some think, more important than medicines. Extreme care is necessary during the periodic attacks of soreness and rawness of the mouth and indigestion. The most important drug used is arsenic, usually given hypodermatically in the form of atoxyl, or sodium cacodylate, and asphenamine. An overdose is indicated by coryza, puffy eyelids or an irritable stomach. The best treatment of the past has been repeated blood transfusions. The greatest improvement, however, sometimes almost marvelous improvement, follows a diet containing large amounts of liver, either fresh, cooked, dried and powdered, or in the form of an extract. Just how lasting the splendid results of this very recent treatment will be we cannot yet tell.

In the past the course of these cases has been up and down. Some patients failed rapidly until death; others seemed to become entirely well and then relapsed. Some of our patients have come to the clinic three or four times. Practically never has the patient been cured. One thing is almost certain, that this anæmia is a late stage of a disease which in some cases has progressed for years before the blood changes appear. Certain symptoms of disease of the spinal cord and of the gastrointestinal canal have suggested the diagnosis of pernicious anæmia even nine years before any anæmia has developed. Surely it is during these earlier years that we should try to cure the disease.

It is very interesting to see how little difference in the symptoms the blood count makes. The symptoms do not seem to depend at all on the number of red cells in the blood.

On the contrary, the low blood count itself is only one of the symptoms of the disease. The girl with chlorosis, hardly able to walk because of the shortness of breath and weakness, may have 4,000,000 cells per cubic millimeter. A man with pernicious anæmia may lead a very active life and yet have a count of only 1,000,000 or lower and seek treatment for some minor symptoms, as gastric indigestion. His symptoms even when extreme may so improve that he is confident he is well, though his count has scarcely risen.

Leukæmia is a word which literally means white blood. The disease is so named because in extreme cases the leucocytes sometimes almost or quite equal the red cells in number and therefore the blood looks milky. In such cases there is of course a marked anæmia (reduction in the number of red blood cells) for the leucocyte count is very seldom above 1,000,000. As a rule it is not over 500,000 per cmm.

Splenomyelogenous leukæmia is a disease of the bone marrow which affects also the spleen and the lymph nodes. These become enlarged, and the majority of patients apply for treatment because they discover a "tumor" in the abdomen; this tumor is the big spleen. The general symptoms felt by such patients are very like those of pernicious anæmia.

Early in leukæmia **the red blood cells** may be normal in number, but usually, and always **later,** they **are diminished,** sometimes greatly. The leucocytes may vary from normal to even 1,500,000. But **the total number of white cells is not** the important sign. If it were we could not separate leukæmia from many cases of leucocytosis. The important point is that in leukæmia unripe leucocytes, i.e. **myelocytes** (see page 63), **are found in the blood.** These young forms of the granular cells with round nuclei, which are never present in normal blood, are in this disease found in large numbers. One finds also many **nucleated red blood cells,** especially megaloblasts. Even when the total count of leucocytes is about normal the constant presence of these abnormal cells will give us the diagnosis.

The best **treatment** of this form of leukæmia is to expose

the spleen, some say the long bones also, to the X-ray. By this means we can keep a patient in fairly good health for years. Radium is often used over the spleen. **Benzol** taken by mouth, if carefully given, may help for a time at least, for the reason that, just as in too large doses it destroys the normal bone marrow, (see p. 69), so in smaller doses it will restrain the diseased marrow.

Lymphatic leukæmia resembles the myelogenous form in its general symptoms, though in this disease it is the lymph nodes which are enlarged, sometimes over the entire body, and forming masses the size of the fist in the neck, axillæ, groin, etc. The spleen is not often much enlarged. The blood, however, looks very different. In the lymphatic form also, the total count of leucocytes is high, but in these cases **the increase** is not of the granular leucocytes but **of the small and large mononuclear non-granular cells.** One may, indeed, fail to find a single cell with granules. The white count seldom runs above 200,000 per cubic millimeter. Sometimes it is the small mononuclears only which are increased, sometimes cells like them but larger. These are, we think the very young forms of small mononuclears, and they also, like the granular myelocytes, occur normally only in the bone marrow. In this form of leukæmia the red-cell count is usually lower than in the myelogenous form. Few nucleated reds are found.

Patients with this form of leukæmia are especially liable to have **hæmorrhages** under the skin, from the mucous membranes, etc.

Acute leukæmia is a very severe, quickly fatal form of leukæmia, so rapid in its development that the disease has no time to enlarge the spleen, lymph nodes, and liver. The bone marrow alone becomes diseased. The leucocyte count is often very high, and the large mononuclear non-granular cells are the ones especially numerous. This disease is interesting because it is so often diagnosticated as diphtheria, scurvy, purpura, typhoid fever, pernicious anæmia, etc.; and

yet, if one examines the blood, he can often recognize the leukæmia at a glance.

Hodgkin's disease, in its early stages, resembles lymphatic leukæmia so closely in every way except the blood picture that for years it was called pseudo-leukæmia. In Hodgkin's disease, however, the leucocyte count is practically normal; and some cases of lymphatic leukæmia begin with, and may have for some weeks, a normal blood picture.

Hodgkin's disease begins with enlargement of the lymph nodes, usually on only one side of the neck. These increase in size until they form a conspicuous tumor. The individual glands in this mass are always firm and remain discrete; that is, they do not soften and do not fuse. Soon the glands of another region, usually of the other side of the neck, begin to enlarge in the same way; then perhaps those in one or both of the axillæ, then, months or years later, those in the groin. Glands in the chest (visible on the X-ray plates) and also those in the abdomen may form large masses. There is no definite order in which these masses of glands enlarge, but it is important in diagnosis that, as a rule, it is in only one region at a time, and that the masses which they form are conspicuously large. If we remove one of the enlarged glands and examine it under the microscope, we can from this alone make a definite diagnosis of the disease. This is very fortunate, especially in early cases, otherwise a case of lymphatic leukæmia or of some form of sarcoma of these glands (especially lymphosarcoma) might confuse us. In lymphatic leukæmia, however, the nodes over the entire body enlarge at about the same time and the single nodes remain separate; also, the masses they form are seldom as conspicuous as in Hodgkin's disease.

Hodgkin's disease affects young men especially. It is progressive in its course and finally fatal. This disease early causes a definite anæmia, a slight loss of strength, and other evidences suggesting a malignancy. The cause we do not know, though it usually follows an infection, especially ton-

sillitis. In itself it resembles an infection; in fact organisms have been discovered which may be important in its causation. The treatment would be the surgical removal of the enlarged glands if we could perform the operation early enough. The cases improve temporarily and considerably under X-ray therapy, but this improvement is not permanent. Arsenic helps somewhat in the treatment.

FIG. 25.—Hodgkin's Disease.

Other forms of adenitis, by which we mean great enlargement of the lymph nodes, may be mentioned here. Infected tonsils, teeth, and nose can cause pyogenic infection and great enlargement of the cervical lymph nodes, which later may soften and give rise to a subcutaneous abscess.

One of the commonest forms of **adenitis** is that due to **tuberculosis.** This occurs especially during childhood, and oftenest affects the lymph nodes of the neck, usually those

of one side but sometimes those of both. These enlarged tuberculous glands may become conspicuous masses which are sore when palpated. The glands, at first firm, tend to soften and become matted together, so that it is hard to feel by palpation the individual glands. In these soft glands develop abscesses which, discharging through the skin, form running sores, sometimes several, sometimes one, on the side of the neck. This condition is called "scrofula." Children with scrofulous glands are usually thin and sick-looking and are very likely to suffer later from tuberculosis of the lungs.

Glandular fever (page 509) is another disease with great enlargement of the lymph nodes, especially those of the neck.

Syphilis also causes enlargement of the glands of the neck, which remain firm and painless but are usually much smaller than in the above condition and must be found by palpation.

Hæmophilia is a very interesting but rare condition which occurs in men, almost never in women. The blood of these men seems, sometimes at least, unable to clot, and they are called "bleeders." Whenever they are struck a large bruise appears, and any small nick in the skin may cause a hæmorrhage that is really serious, since the blood may ooze from this cut for days. Should a surgeon inadvertently operate on such a case, he would doubtless kill his patient. These men are careful never to shave, for fear they may cut themselves even a trifle. They may bleed to death from the nose, or from the mouth after a tooth has been pulled.

It is interesting to note that, while **hæmophilia occurs almost exclusively in men,** they **inherit it from their mothers,** not from their fathers. For instance, if Mr. A is a bleeder, neither his sons nor his daughters will be bleeders, but his **daughters' sons** will be, not his sons' sons. That is, the disease is transmitted only through the women in the family, and yet they themselves are not bleeders. For the treatment of this condition, as in all diseases with severe hæmorrhage, like purpura, etc., we do a tranfusion, if possible, and give

the patient some form of calcium. Calcium is necessary in the coagulation of blood, and if we prescribe it to a patient whose blood clots too slowly the blood will soon clot much faster. The best form of calcium to give is the lactate, 30 grains three times a day.

CHAPTER VI

The Organs of Circulation and Their Diseases

The Mechanics of Circulation

The circulation of the blood will now occupy our attention. The blood is pumped throughout the body in a system of closed tubes, the blood vessels, which are the arteries, capillaries, and veins. It is pumped through two systems of vessels; the systemic system, extending from the heart through all the body and back to the heart; and the pulmonic system, extending from the heart through the lungs, and back to the heart. If we trace a blood corpuscle in its journey (Fig. 26), starting at the aortic valve, *n*, we find that when it leaves the heart it enters the aorta, *a*, passes into one of the many branches of the aorta and thence through smaller and smaller arteries, until the artery becomes a capillary—in a muscle, for example. The corpuscle passes through a fine capillary, *b*, of this muscle and enters a small vein, *o*, whence it moves on through larger and larger veins till it finally enters the vena cava, *c*, which carries it to the right auricle, *d*, of the heart. The right auricle pumps it into the right ventricle, *e*; the right ventricle pumps it through the pulmonic artery, *p*, into the lungs, *f*. It passes through the capillaries of the lungs into the pulmonic veins, *q*, then down to the left auricle, *g*, which pumps it into the left ventricle, *h*, and thence through the aortic valve, the starting-point. If we should trace another corpuscle it might take the following course. From the left ventricle it would pass into the aorta, then through the mesenteric artery, *m*, perhaps to the capillaries of the intestines, *i*, through the mesenteric vein to the portal vein, *j*, which would carry it into the liver, then through the capillaries of the liver, *k*, into the hepatic vein, *l*, through this into the vena cava, to the right side of the heart, and then as before. In this case it would have passed through three sets of capillaries—those of the intes-

tine, the liver, and the lungs. This is known as the "portal circulation."

We see, therefore, that we really have **two hearts,** which do not directly communicate with each other (Fig. 27). **The**

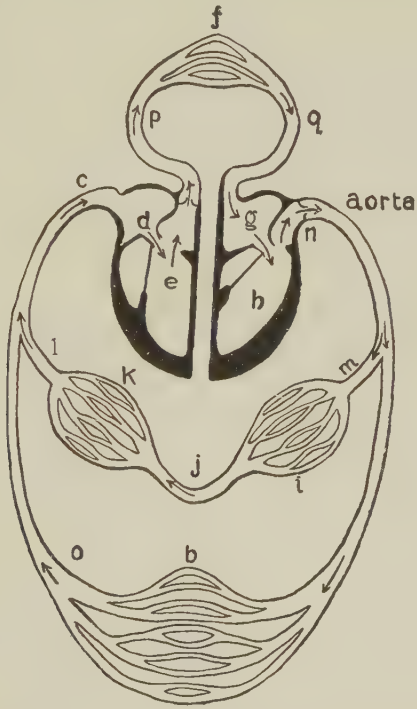


FIG. 26.—Diagram of the circulation of the blood. *a,* aorta; *b,* the capillaries of the systemic circulation (*e.g.,* muscles, brain); *c,* vena cava; *d,* right auricle; *e,* right ventricle; *f,* capillaries of the lungs; *g,* left auricle; *h,* left ventricle; *i,* capillaries of the stomach and intestine; *j,* the portal vein; *k,* capillaries of the liver; *l,* hepatic vein; *m,* mesenteric artery; *n,* aortic valves; *o,* a vein anywhere in the body except the lungs or abdominal viscera; *p,* pulmonic artery; *g,* pulmonic vein.

right heart, *A,* consisting of auricle and ventricle, pumps the blood through the lungs; **the left heart,** *B,* auricle and ventricle, pumps the blood through the body. These two hearts are bound together into one, but their union does not imply any direct communication as far as the blood current is concerned. That the blood may always be forced in the right

direction, the heart is provided with valves. When the right ventricle contracts, the tricuspid value, *a*, prevents any back-flow to the right auricle. When the contraction is over and the ventricle is limp, the pulmonic valve, *c*, prevents the blood from flowing back into the ventricle. In the same way on the left side, the mitral valve, *b*, prevents the back-flow into the left auricle, and the aortic valve, *d*, prevents the back-flow into the left ventricle.

We speak of the contraction of the heart as its **systole**; the period during which it is limp and resting is called its

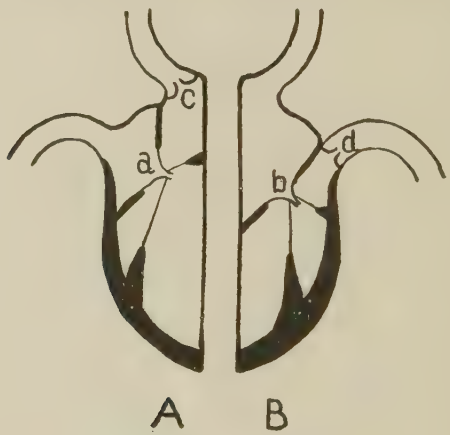


FIG. 27.—Diagram of the two hearts. *A*, the right heart; *B*, the left heart. *a*, tricuspid valve; *b*, mitral valve; *c*, pulmonic wave; *d*, aortic valve.

diastole. The valves are membranes of wonderful strength, yet almost as thin as heavy paper. The edges of the mitral and tricuspid valves are anchored by the chordæ tendineæ, which are fine but very strong threads.

The heart is a wonderfully powerful little **pump**. It **contracts** about seventy times a minute, with each contraction throwing forcefully about 100 c.c. of blood from each ventricle. It must by each beat throw exactly the same amount from each ventricle. If it did not, the blood would accumulate in either the pulmonary or the systemic circulation, and a difference of only one drop per beat would mean about 300

c.c. per hour. Since the only function of the right side is to pump the blood through the lungs, its walls need not be nearly so strong as those of the left side, which has the much harder task of forcing the blood to the most distant parts of the body. The 100 c.c. of blood are expelled into the artery with such force that, if an artery is cut the blood will spurt six or more feet. The sudden gush of blood from the ventricle sends a "pulse wave," as we call it, down through the arteries even to the capillaries.

The little **corpuscle** we were following **travels** through the arteries at the rate of about one yard in three and a half seconds, or about one mile per hour. The capillaries are very narrow, but their combined width so much exceeds that of the arteries of which they are the branches that the blood flows through them slowly, only about one yard per hour; but they are only about ½ mm. long, and so it takes only about one second for the cell to pass through them into the veins. Here the corpuscle travels in a steady stream, the speed of which increases as it approaches the heart. The large veins are not nearly equal in calibre to the sum total of the smaller veins which feed them, and, just as the water in the narrow part of a river flows more rapidly than it does through the meadows which that river drains, the blood flows faster and faster as it approaches the heart. It has been reckoned that the total capillary "bed" (as one speaks of the "bed" of a river) is about eight hundred times as wide as the aorta which feeds it.

The **whole circuit** of a corpuscle, from the aortic valve around the body and back to that valve, requires only about twenty-three seconds. One fifth of this time is required for the circuit from the heart through the lungs and back again to the heart. To push this corpuscle through the whole circuit requires twenty-seven heartbeats; and, since the heart expels about 100 c.c. of blood with each beat, every drop of blood in the body could pass through the heart in fifty-three beats, or in about forty-five seconds.

The work necessary to accomplish the task of moving

the blood is enormous. The heart is about the size of a man's fist. It works day and night, and each hour it accomplishes as much work as do the strong muscles of the legs of a man weighing 140 pounds who every hour climbs a staircase sixty feet high. But the heart does not work all the time. We find that it rests twelve hours a day, if we add together the time of its pauses between its beats, during each of which it is perfectly limp, and so completely tired out that at the beginning of this pause a strong electric current could not arouse it. For three hours a day it does light work, that of pumping the blood from auricles into ventricles, a relatively easy task. It works hard for nine hours a day, the sum of the moments consumed by the ventricle in forcibly pumping the blood into the arteries.

The arteries are always overfilled, and their walls are considerably stretched by the large amount of blood within them. Since the arteries are very elastic and are distended, their elastic recoil is, between heartbeats, constantly forcing the blood on into the small arteries and capillaries and thus keeping up a continuous circulation.

The blood vessels through which the blood circulates may be divided into three classes: the arteries, veins, and capillaries. **The arteries** begin at the heart with the aorta and carry the blood to all parts of the body, dividing, as they go, into smaller and smaller branches until they reach the capillaries. The walls of the arteries must be very strong, since the heartbeats keep the blood in them under such high pressure. Their walls (Fig. 18) consist of three layers. The inner layer is the so-called **intima**, *A*, a thin membrane of flat cells, which furnishes a surface suitable for contact with blood flowing through the artery. Outside the intima is a thick **media**, *B*, which consists of muscle fibres mingled with strands of strong elastic tissue. This muscle tissue is of the so-called "involuntary" type; that is, we cannot control it by our will. It strikingly differs in appearance from the voluntary muscle, of our arms, for example, and differs from it also in that it can without tiring remain contracted

practically for its lifetime. The media is the layer which makes the artery strong, and able to stand the high pressure within it. Outside this is a thick but rather weak layer of connective tissue, the **adventitia,** *C*, which binds the artery to the structures through which it passes, and which serves as packing to fill the crevices around the arteries. The muscle of the media is under nervous control. When a person blushes, for instance, the nervous impulse from the brain allows the media of the blood vessels of the cheek to relax, and hence these arteries dilate; that is, their lumens become much larger, and much more blood rushes to the capillaries. If the skin is chilled a person becomes pale, because the media

FIG. 28.—Diagram of an artery, capillary and vein. Note how the walls of the artery "thin out" until the wall of the capillary is only the intima, a single layer of cells. (Of course the transitions from artery to capillary, etc., are actually very much more gradual.) Magnified 300 times.

contracts, and reduces the lumen, sometimes to one half its previous size.

As the arteries (Fig. 28) by dividing become smaller and smaller, their media and adventitia become thinner and thinner, until the walls of the smallest arteries consist of the intima and a very thin layer of muscle fibres. Here begin the **capillaries,** which are about 1-32 of an inch long and just wide enough for one red corpuscle to squeeze through. At their other ends begin the smallest **veins.** The intima of these has around it a very thin layer of media. As the veins unite to form larger and larger veins, the media becomes thicker and an adventitia is added; but the walls of even the largest veins are thin compared with those of an artery of the same size, because the blood within the veins is under much less pressure.

If one should be asked what part of this circulatory sys-

tem—the heart, aorta, small arteries, capillaries, veins, and, finally, the largest vein (vena cava)—was the most important, he might at first think of the heart and the larger vessels; but in fact **the most important part** of the whole system is the tiny **capillary,** which is only about 1-32 of an inch in length. It is in order that the blood may pass through those tiny tubes that the whole "cardio-vascular system" exists.

The function of the blood is to nourish the body. The nourishment within the blood vessels cannot pass through the walls of the arteries and veins but can pass through the single layer of intima cells, *a,* of the capillaries (Fig. 29). In these tiny vessels the blood flows very slowly. The plasma soaks through their walls, furnishing new food to the tissue lymph, and then to the tissue cells, *b,* taking up much of the waste matter which these cells have excreted. It is here that the oxygen leaves the red blood cells and goes to the tissues, and the carbon dioxide leaves the cells and goes to the plasma. The importance of the capillary may become clearer if one thinks of the stations of the railroads which pass through any city. Suppose there were no stations, that the inhabitants of that city were starving, and that freight trains full of food, enough to furnish the citizens in great abundance, were thundering through the city on the main tracks every ten minutes. The inhabitants of that city would continue to starve unless these trains should stop and the food should be unloaded, so that the citizens could get it. In a similar way the heart might pump the blood through the arteries into the veins and back to the heart in abundant volume, but the tissues would starve if it were not possible for the food to be unloaded in the capillaries, the stations.

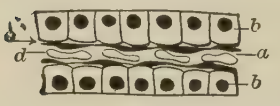


FIG. 29.—A capillary and the cells which it furnishes with food and oxygen and whose excreta it removes. *b,* tissue cells; *c,* lymph space between cells and capillary wall. *d,* red blood cells represented as squeezing through the narrow capillary. (Magnified 600 times.)

The lymphatic system consists of another set of tubes which ramify throughout most of the body. These start as capillaries that drain the tissue spaces (Fig. 29, *c*), then unite to form

the lymph vessels which pass through the lymph nodes, and then finally empty into the large "thoracic duct", which empties into the large vein on the left side of the neck. Such certainly is true of the lymphatic vessels of the small bowel, for these maintain a steady flow of digested fatty food (chyle) from the intestinal mucosa to the thoracic duct. They supply by far the most of its contents. Lymphatic vessels are found in nearly all parts of the body, but they seem to converge into clusters of lymph nodes, which are found at definite points (the neck, axillæ, groin, etc.); and while finally all reach the thoracic duct, there is normally very little real flow. Except in the abdomen the most of the tissue lymph seems to flow out of the blood capillaries and back into them. Indeed, one of the chief values of the lymph vessels seems to be potential, and most evident when the tissues are diseased, since then they remove from the infected area the toxin of the disease and the products of infection. This explains the red lines which run up the arm in a case of streptococcus infection of one finger tip.

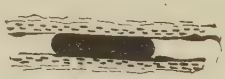


Fig. 30.—A small artery plugged by a thrombus.

Diseases of the Blood Vessels

Located along the course of these lymph vessels are the **lymph nodes,** the filters of the lymph flow, and, in part at least, the source of some of the blood lymphocytes. These lymph nodes swell as a result of infections within their area of drainage, and also as a result of Hodgkin's disease, tuberculosis, and cancer. Like filters these nodes seem to try to stop the progress of the infection or cancer, and they usually succeed, but in severe cases of the former and in all cases of the latter the gland itself soon loses its power to protect and develops as a new center from which the disease spreads.

A thrombus is a blood clot which forms inside a blood vessel. When one is present that vessel is said to be thrombosed, and the condition is called **thrombosis.** The clot may be small and cling to the vessel's wall, or it may completely plug the vessel (Fig. 30), in which case it prevents the blood

from passing any further along that course. We think that either the wall must be injured or germs must have settled in it and started an inflammation there before the blood could clot at that point; for the normal intima should not allow this to occur. The first result of this injury is the deposition there of masses of platelets which by disintegrating liberate substances which cause the formation of the fibrin, which entangles blood cells and soon forms a clot strongly resembling those mentioned on page 48.

Fig. 32.

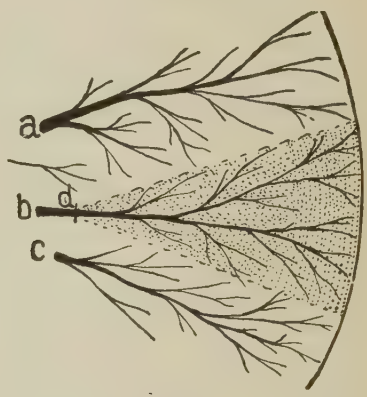


Fig. 31.

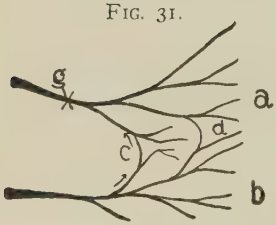


Fig. 31.—Two anastomosing arteries. *a*, and *b*, are two small arteries which are connected by two small communicating branches or "anastomoses," *c*, and *d*. If *a* should become plugged at *g*, the circulation through *a* would not stop since the blood would flow from *b* through *c* and *d* into the branches of *a*.

Fig. 32.—End-arteries, and an infarct. *a*, *b*, and *c* are three small arteries which have no communicating branches. If *b* becomes plugged at *d* then all the tissue supplied with nourishment by *b* must die. This dead area is an "infarct." (The dotted triangle.)

The **results of a thrombus** will depend entirely on the nature of the circulation where it occurs. If it occludes, or plugs up, a vein, the blood will be dammed back, but the blood can reach the heart along some other channel. The arteries, however, are different. Of these there are two kinds in the body—those which **anastomose** (Fig. 31), that is, send branches across to neighboring arteries; and those which do not. The latter are termed **end-arteries.** (Fig. 32.) If an end-artery, *b*, becomes plugged, as at *d*, all circulation along that channel stops, and the tissue which that artery

feeds must die. If it is not an end-artery (Fig. 33), then the plug, *y*, may make little trouble, for the arteries in the neighborhood, *b*, which are not plugged will send some blood through the short communicating or anastomosing branches, *c*, to fill the artery beyond the plug and so keep up its circulation. Hence the tissue which the plugged artery feeds will receive more or less nourishment. At once these small anastomosing arteries begin to grow larger and larger until they are able to carry all the blood necessary. Then, we say, a sufficient **collateral arterial circulation** has been established. There are practically no end-veins and the anastomosing branches between veins are so numerous that to establish a sufficient collateral **venous circulation** is easy; hence a venous thrombus does relatively little damage. It is a very interesting fact, and one hard to explain, that the most important organs of our body—the heart, brains, kidneys, spleen, intestine have either end-arteries or arteries which anastomose so little (e.g. the heart) that they practically are end-arteries, since if any of their branches become occluded a certain amount of tissue will die. Since the arteries as a rule flow from the centre of the periphery of an organ and branch like a tree, it follows that the area which dies after the closure of an end-artery will be wedge-shaped, with its base on the surface of the organ. These dead areas are known as "infarcts" (Fig. 32).

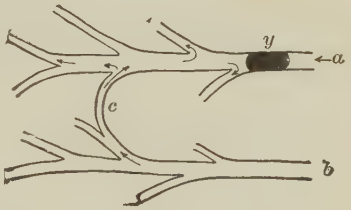


FIG. 33.—Anastomosing arteries. *a* and *b* are connected by an anastomotic branch *c*. If *a* is plugged by the thrombus *y* the circulation in *a* will be maintained by *b*.

An embolus is a thrombus which has become dislodged, that is, which breaks loose and is swept on in the blood current through the blood vessels until it comes to one which it cannot pass through, and this it plugs tightly (Fig. 34). This process is called **embolism.** The majority of such thrombi form on the walls of the cavities of the heart, especially on the edges of the heart valve. An embolus from a

vein or from the right heart will travel to the lungs and occlude a branch of the pulmonary artery. One from the pulmonic veins or the left heart or a large artery will plug a small artery. Where the embolus stops it becomes again a thrombus. Its results will depend on the artery which it happens to plug. If this is an artery with sufficient anastomosing branches, there may be no result; but if it is an end-artery, the result will be an infarct. If, however, the embolus contains germs, the spot where that embolus lodges will be a centre of infection and perhaps the starting-point of an abscess. It is an interesting and an inexplicable fact that those organs in which an embolus can do the most damage are the very ones in which the embolus is most likely to lodge. It often goes to the heart muscle through one of the coronary arteries, which leave the aorta just behind the aortic valve. The result in this case is the sudden death of a part of the heart wall and, unless this is a very small part, the paralysis of the whole heart. Another favorite spot for an embolus to strike is the brain, since the vessels to this form the straightest path for an embolus leaving the heart; and, unfortunately, of the whole brain that part most often hit is the one which ought to be most carefully guarded, the left motor area, since paralysis of the right side of the body with speech disturbance (aphasia) is the result. The spleen and the intestine also are favorite places for the embolus to strike. In the case of the intestine, if the embolus is at all large, death is almost certain to follow. Emboli to the kidney or spleen always produce infarcts, but not with so serious results.

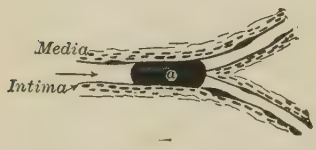


FIG. 34.—An embolus plugging a small artery. The embolus *a* was forced along by the blood stream until it reached a bifurcation of the vessel; here it stops.

Arteriosclerosis ("hardening of the arteries") is the disease which indirectly kills the majority of men. The media is the most important layer of the vessel wall and the one most sensitive to injury. Any agent which attacks an artery

will do most injury to this layer. Suppose that in a small area of the artery's wall some muscle fibres should be killed by a poison, by infection, or by the closing of the tiny blood vessels which nourish that part of this artery's wall (Fig. 35). At that point the media would become thin and weak. Either the wall would burst at that point because of high pressure of the blood current within, as happens in a water main; or, since the wall is elastic, it would "balloon out," forming a blood tumor (aneurism). The body protects itself against such accidents by increasing the thickness of the intima opposite each weak point in the media, d, and hence, as the media weakens, the intima becomes stronger, and a small, thick plaque of intima, d, forms here. But this plaque is nothing but ordinary, weak, scar tissue, which has very little vitality and soon dies. Lime salts are then deposited in the dead tissue, and a small fragment of hard shell is there formed; or this dead tissue will slough out and leave an ulcer in the arterial wall. This **thickening of the arteries' walls is known** as **arteriosclerosis.** Sometimes the arteries are thickened along their whole course, sometimes only in certain areas, and sometimes in little spots over wide areas. If an artery has a large number of these plaques along its course it is by no means a normal artery. Then too, these plaques choose as a favorite point for their formation the spot where a small branch artery leaves an artery (Fig. 36, *a*), and hence this branch soon has its mouth partially choked by the plaque of sclerotic tissue.

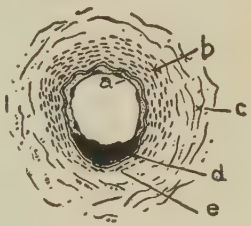


Fig. 35.—Arteriosclerosis of a small artery. *a*, intima; *b*, media; *c*, adventitia; *d*, a mass of thickened intima which strengthens the weak spot *e* of the media.

Fig. 36.—Arteriosclerosis. A small patch of arteriosclerosis *a* is partially occluding the mouth of a small branch artery.

Some of the **results of arteriosclerosis** are easy to understand. Sclerotic arteries are more or less rigid, not elastic, tubes, and so more force is required to pump blood through them. The first result, therefore, is that the heart must work

much harder in order to keep up sufficient circulation. We know that if we exercise any of our muscles they get thicker and stronger. So it is with the heart. Increase its work, and it enlarges, or hypertrophies. But, though larger and stronger, it is not necessarily better than a normal heart; in fact, in some ways it is a less serviceable organ, for reasons which we shall mention later.

The next result of arteriosclerosis may be stated as a law in pathology, that whenever a tissue receives insufficient food, it becomes weaker or dies, and, if it dies, its place is taken by a tissue which requires less food. That tissue which requires the least food, the one which usually replaces starved cells, is fibrous, or scar, tissue, a tissue which cannot perform any of the specific functions of the cells which it replaces. Scar tissue also contracts (hence, the "scars") and causes a shrinking of the diseased organ. Those tissues which perform the most active functions require the most food and are very sensitive to any reduction in this food supply. To illustrate: a person with general arteriosclerosis will sooner or later have heart walls in which much heart-muscle fibre is replaced by inert scar tissue, the result of arteriosclerosis of the coronary arteries, those vessels which supply the heart's wall itself with blood. Because of the arteriosclerosis of the whole body the work of the heart in this case would be increased, thus necessitating an increase in its strength, but, since this same disease is also in its own walls, it cannot respond adequately. A kidney fed by sclerotic vessels may shrink to about one half its normal size; a liver may be of about one half scar tissue; and in the brain may be evidence of such atrophy.

The causes of marked degrees of arteriosclerosis may be reduced to heredity, wear and tear on the arteries, infections, and poisons; and the degree of the sclerosis will in large measure depend on the quality of the material of which the vessels were originally built. Advancing age is usually considered the most common cause of all and sclerotic arteries are considered almost as normal for elderly persons as white hair. This is scarcely the case. While it is true that, the

older a man lives, the more opportunities he has had for developing arteriosclerosis, yet in every case in which it develops it should be considered abnormal, and many an elderly man has soft arteries. Some persons develop arteriosclerosis too easily; in fact, there are some families all or many of whose members have arteries which grow old too soon. These persons were born with "poor rubber in their tubes," and hence their blood vessels, and so their whole bodies, show age more rapidly than do those of members of other families. Since the health of the whole body depends to a considerable degree on the vascular system, there is much truth in the old adage that a "man is as old as his arteries"; that is, some persons at forty can boast of little better health than most men at seventy.

Excessive "wear and tear" is thought to be one of the most important causes of early sclerosis, and this is ascribed, in part at least, to abnormally high blood pressure, to hard muscular work, to overeating, and especially to long continued worry. Yet we see many who work hard, eat heavily, worry, and do hard physical work, but whose arteries are still soft. Those with high blood pressure are not so fortunate, nor are those with Bright's disease, whether they have high blood pressure or not. The fact is that in most of the cases of arteriosclerosis, perhaps in all, some more definite cause than "work and worry" is necessary, such as **chronic infections** of the nasal sinuses, tonsils, teeth, etc.; while others owe their thickened arteries to **poisons** such as **alcohol** or lead, and more owe them to the poison of **certain fevers**, such as gout or typhoid fever or—and especially—lues, etc.

The treatment of arteriosclerosis is very important, since, as we have said, arteriosclerosis, because of the brain, heart, and kidney diseases which it causes, indirectly kills the majority of adult men. While any damage already done to an artery cannot be repaired, yet the process producing it surely can be slowed down or stopped. With the exception of luetic arteritis, which may be quite local, arterial disease when present is quite universal throughout the body, involving always

those most important systems, the myocardial and renal vessels. The term **arteriosclerosis** is therefore now often replaced by the more correct term **cardio-vascular-renal disease.** All local infections in tonsils, teeth, nasal sinuses, appendix, and pelvis, should, as far as is possible, be eliminated, and lues, if present, thoroughly treated. The climate particularly is important, and these patients should if possible spend their winters in the South, while those who remain at home should be protected in every way from chilling. Physical and electrical therapies, warm baths, etc., have some value, since they produce muscular relaxation.

The skin especially deserves attention since its condition indirectly affects that of the kidneys and bowels. It should be kept warm and moist by warm baths. The patient should drink sufficient water and avoid constipation. Old people in particular stand exposure to cold badly, because of its effect on the skin and therefore on the renal function. In caring for them one protects them from cold air, cold baths, or any radical change in their customary clothing and surroundings. Any exposure to cold should be met by warm clothing, warm foot baths, and hot drinks. If in bed and not allowed to wear flannel underwear they should have extra blankets, hot-water bags, and frequent massage to the limbs to restore the circulation and prevent cold, cramps, etc. All excesses in exercise, food, drink, and habits should be avoided.

Constipation especially should be combated, using salines and the stimulating cathartics. **The diet** should consist chiefly of milk, fresh fruit, cereals, eggs, little meat, and entirely avoiding kidney, liver, sweetbreads, brain (purin rich foods) and all condiments. When the arteries of the brain are affected the danger of apoplexy must always be remembered, and all causes of worry, excitement, anger, or irritation avoided, since these greatly increase the cerebral blood supply. Slight petty causes of irritation, which particularly upset the patient, should be avoided. The bromids, luminal, amytal or morphin may be necessary to relieve the pain and to quiet the patient.

Of drugs, a useful one is potassium iodide, taken in milk after meals, and in doses of about 10 grains, three times a day. Nitroglycerin is often used, but its value is rather doubtful, unless there are pains in the chest which it will relieve. Of this we give one drop of one per cent solution three times a day on the first day, then increase the dose if necessary, stopping when a headache and throbbing in the head begins.

Aneurism. As you already know, the arteries are elastic tubes, through which flows a fluid under high pressure.

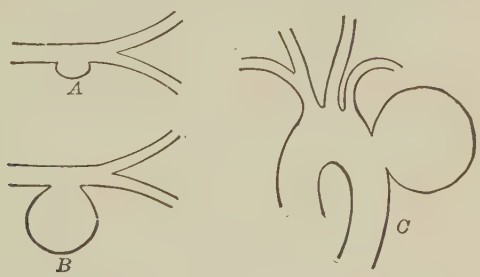


FIG. 37.—Saccular aneurisms. *A*, a small saccular aneurism; *B*, a later stage of *A*. *C*, saccular aneurism of the arch of the aorta. A very common and a huge form. These may be several inches in diameter.

If the wall becomes weak at any point and does not burst, and the weak spot is not rapidly enough strengthened by the thickening of the intima, the wall will be forced out, very much as a rubber bag is inflated, by the pressure of the blood within, and a large pulsating blood tumor be formed. This is called an **aneurism.** If the weak spot is very limited in extent, so that the aneurism projects from one side of the artery, we have a **saccular aneurism** (Fig. 37, *A, B, C*); if the whole artery is dilated, we have a **fusiform aneurism** (Fig. 38, *A*, *B*). The body from the first protects itself by forming new tissue around this tumor, which in this way soon gains a thick wall, while within the sac the blood may clot thus effecting a cure. The great majority of the aneurisms rupture, causing sudden death, though through a small rupture the blood may work its way along between other

organs, scar tissue meanwhile forming a wall around the tumor to limit the escaping blood. This is known as a **dissecting aneurism.**

The symptoms of aneurism are those of tumor plus the pulsation due to the pulse wave of the blood inside.

Our treatment in the case of a large aneurism of the aorta is to aid the blood within the sac to clot solidly. The patient is put to bed and kept as completely at rest as possible in order to reduce the number of heart beats, by fifteen per minute if possible, and thus in some degree to quiet the blood within the tumor. In this way the heart is spared nearly five of every twenty-four hours' of its work. The patient also is kept on a very limited diet that his blood pressure may be low. Or about ten feet of wire are inserted in the tumor and an electric current run in, in the hope that the foreign body and the electric current will stimulate coagulation. This succeeds with some saccular aneurisms. The aneurisms of small arteries are more easily cured by partially or wholly occluding the vessels, but, since most aneurisms are on the aorta or its big branches, this method can seldom be used.

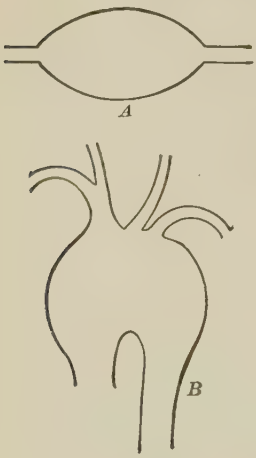


FIG. 38.—Fusiform aneurisms. *A*, a fusiform aneurism of a small artery. *B*, fusiform aneurism of the arch of the aorta, the commonest form. This is also called "diffuse aneurism," or "dilatation of the arch."

Large aneurisms are not very common, but **minute (or miliary) aneurisms** are exceedingly so. For example, a hæmorrhage from the lungs in tuberculosis, when it occurs late in the disease, is nearly always due to the rupture of a miliary aneurism in the wall of an exposed pulmonary artery. The same is true of a hæmorrhage from a gastric ulcer, it is the small aneurism of the exposed vessel which ruptures. Hæmorrhage into the brain ("apoplexy") has a similar cause. In nearly all of these cases the artery was first injured at one point by the disease and there developed a tiny saccular

aneurism, which later ruptures. It is one of the mysteries of our life that that cerebral artery which most often ruptures is the one which by rupturing does the most harm. This tiny artery, called the **artery of cerebral hæmorrhage** (Charcot), is the one which feeds the internal capsule of the left hemisphere of the brain. If one should try to cripple the whole of the telephone system of one section of a city, he might go about it in two ways. He might pass through the streets, cutting the wires wherever he found them, but this would take a long time. He could go to the central station and there with one blow sever every wire at the point where they are all bound together in one bundle as they leave the station before scattering over several square miles. So it is in the brain. The nerve fibres leave the wide area of the cortex. They then collect together in one small bundle at the base of the brain, known as the internal capsule, pass into the spinal cord, and are all distributed to the whole of the opposite side of the body. Hence it is that a tiny hæmorrhage from the artery feeding the internal capsule can paralyze almost every muscle on the opposite side of the body. Our speech is controlled from the left side of the brain and from an area adjoining the motor area, and hence the person paralyzed on the right side of the body usually is unable to speak (aphasia). It is hard to understand why this most important point should be the most vulnerable for an aneurism.

Diseases of the Heart

Anatomy. To understand **valvular heart disease** we should remember that we have two hearts in one (Fig. 27) —a right, *A*, and a left, *B*—each with an auricle and a ventricle. These two hearts, though united in one, have no direct communication with each other. The right heart pumps the blood through the lungs to the left heart—**the lesser circulation**—while the left heart pumps it through the body and around to the right heart—**the greater, or systemic,**

circulation. Both auricles contract at the same time, the right forcing the blood through the tricuspid valve, *a*, into the right ventricle, and the left forcing it through the mitral valve, *b*, into the left ventricle. The ventricles when full contract forcibly. The pressure thus created in the ventricles first closes the tricuspid and mitral valves, then, when on the right side it is greater than the pressure in the pulmonic artery, and when on the left side it is greater than that in the aorta, it forces the pulmonic valve, *c*, and aortic valve, *d*, open, and blood is forced into these two arteries. Each beat ejects about one hundred cubic centimeters of blood from each ventricle. As soon as the ventricles are empty their walls become perfectly limp. Then the blood in the pulmonary artery and in the aorta, in both under considerable pressure, tries

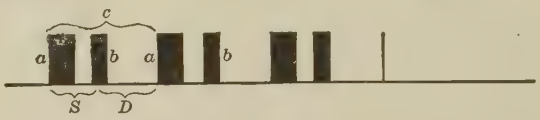


FIG. 39.—A graphic representation of the normal heart sounds. *a*, the first sound; or "lub"; *b*, the second sound or "dub." *c*, one heart beat, of which cycle "*S*" is the systole, (from the beginning of the first to the beginning of the second sound), *D*, the diastole (from the beginning of the second to the beginning of the next first sound).

to rush back into the ventricles but is checked by the pulmonic, *c*, and aortic, *d*, valves.

When the ventricles contract and close the mitral and tricuspid valves, the muscle walls and the valves together make a sound which is best described by the syllable "lub." When the pulmonic and aortic valves close they make a shorter, sharper sound, like the syllable "dub." **The heart sounds** produced by one contraction, or systole, are thus represented by the sounds "lub dub." Graphically they may be represented by Fig. 39. The period from the beginning of the first sound to the beginning of the second is called "systole," that from the beginning of the second to the beginning of the next first, "diastole." In general, however, the word "systole" is used to mean the heartbeat.

The **heart** occupies a **position** indicated in Figs. 40 and 41. Usually one can see the apex of the left ventricle move the

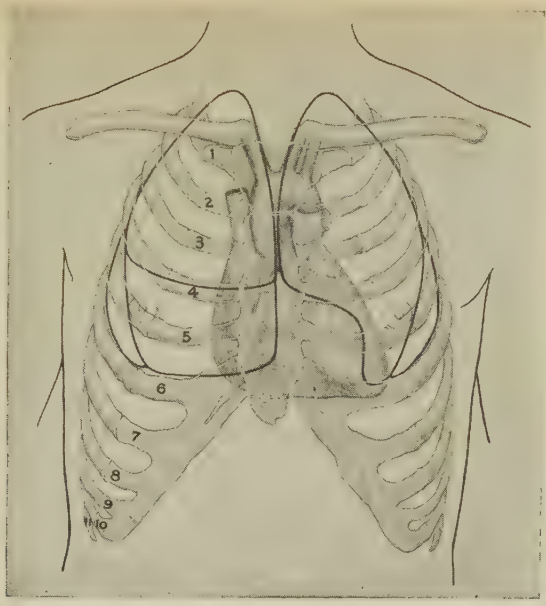


FIG. 40.—Outline of the heart relative to the ribs and lung margins.

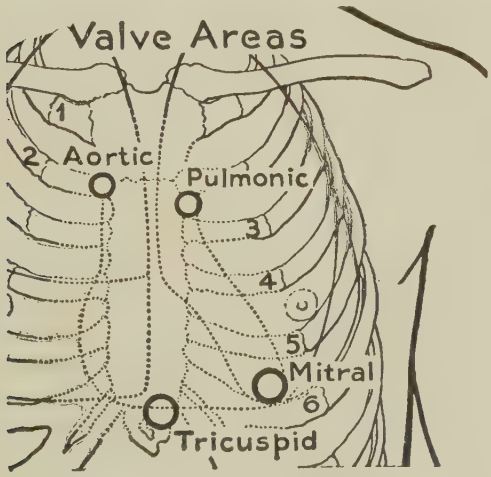


FIG. 41.—Valve areas.

chest wall with each beat at (Fig. 67) *A*, a point just below and median to the left nipple in the fourth or fifth costal interspace. This spot is called the **point of maximum impulse**, or the P. M. I., and this pulsation is called the "apex beat." The areas where the various valve sounds are best heard are here indicated. Thus *A* is called the **mitral area.** The mitral valve is at some distance from *A*, but its sounds are best heard at that point. *B* is the **pulmonic area**; *C*, the **aortic area**; *D*, the **tricuspid area.** These are not the positions of these valves but are the points on the chest wall where their sounds are best heard. (Fig. 41.)

The sudden addition of about one hundred cubic centimeters of blood to the contents of an artery already full stretches its walls still more and starts a wave which travels

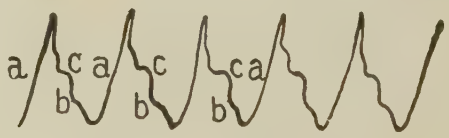


Fig. 42.—A diagram of the pulse waves. (A modified "pulse tracing.") *a*, the ascent of the wave; *b*, the descent, and, *c*, the dicrotic wave.

down the arteries to the capillaries and which is called the **pulse wave** or the **pulse.** If this wave is represented graphically, as by Fig. 42, its ascent, *a*, due to the sudden influx of blood, is sharp; and its descent, *b*, is more gradual, since the blood is slowly squeezed onward into the smaller vessels by the overdistended, elastic arteries. On this descent is seen a small wave, *c*, called the **dicrotic wave,** which is produced by the rebound of the blood column when the aortic valve closes. Normally the pulse is very steady, the beats following each other with great regularity. Even in severe aortic-valve disease the rhythm is regular, but in mitral-valve disease and especially in myocarditis its irregularity is a marked feature of the case.

Physiology. Understanding the anatomy of the heart, we can now consider **the physiology of the heartbeat.** All parts of the heart wall (myocardium) have the ability and

the tendency to contract. Cut the ventricle free from the rest of the heart, and under proper conditions it will beat regularly, about 28 times per minute. All parts of the auricles, however, are even much more ready to beat than are the ventricles, and at a faster rate. But the heart has a **pace maker,** where normally all of its beats start, the sino-auricular node, located just where the superior vena cava enters the right auricle. First this center stimulates the auricles and then the stimulus of their beat passes down to the ventricles through a special bundle of fibres, the **auriculo-ventricular bundle,** called also the bundle of His, and stimulates the ventricles to beat. In this way each cardiac contraction, involving four heart chambers and many separate layers of fibres in each, progresses in an orderly and efficient manner.

The irregularities of the heart's action are due to disturbances of this mechanism which governs the rhythm of the beats. The most common irregularity is caused by **extrasystoles** mentioned on page 34. For example, while the heart is beating regularly, all its beats originating in the sino-auricular node, suddenly some part of the auricle or of the ventricle adds one extra beat. These single beats which do not arise in the sino-auricular node are called extrasystoles. If this extrasystole follows soon after one of the regular beats, the patient may feel only one beat, but with our stethescope we can hear both. Because of this extrasystole the heart cannot respond to the next regular stimulus from the node and will therefore rest quietly until the regular stimulus next in order produces its beat. It is either this unusually long pause or the following beat which, since the heart has had so long a rest, will be unusually strong, of which the patient complains. Some say that the heart stops; some, that it "thumps"; some, that is does both. The reason why the next regular stimulus from the node, after the extra beat, is lost is that it reaches the heart while it is making, or just after it has made, its extra beat. The heart uses up for each beat all the energy it has and so for a brief period

cannot be stimulated either during or immediately after a beat. This period is called its refractory phase. It then slowly recovers its energy for the next beat and responds to stimuli, at first feebly, then more strongly, until it can beat with normal strength. **Extrasystoles may mean** that the heart is injured, but they more often mean that the patient is merely very nervous, smokes too much, or eats too fast.

In another form of irregularity the heart suddenly begins to double or treble the number of its beats per minute, continues at this rate for a while, and then suddenly returns to its normal speed. The change in rate comes suddenly, between two beats. These periods of regular and rapid action of the whole heart, called **paroxysmal tachycardia,** may last for minutes, hours, or even days. Some persons have such periods occasionally all throughout their lives. They feel exhausted while the attack lasts, but they know that it will soon pass. Often the patient has little or no distress, merely shortness of breath and a peculiar feeling of apprehension; sometimes there is a definite sense of fluttering under the sternum; more rarely there is pronounced palpitation; and still more rarely there are severe anginal pains. Sometimes the patient can head off an attack just when it begins, but as a rule, he must lie down and wait for it to pass over. It is really of little serious importance.

Auricular fibrillation is a very serious irregularity of the heart, which develops in about one half of all cases of severe heart disease. In these cases two, three, four, or more parts of the auricle all try at the same time to beat independently, with the result that the auricle merely quivers, that is, fibrillates. This means that the sino-auricular node has entirely lost control of the heart. Each of all these abnormal beats sends its stimulus to the ventricle, which responds as often and as strongly as it can to this confused series of stimuli; but of course the ventricular beats which result are quite irregular both in force and in rhythm, and the heart is unable to maintain an efficient circulation. We often can check auricular fibrillation with digitalis in large doses or, better,

with quinidine sulphate, from two to six grains three times a day.

Auricular flutter, on the other hand, is a different and a more serious condition, present especially in elderly persons. In these cases there is auricular tachycardia, that is, the auricles beat very rapidly but regularly, perhaps three to four hundred times a minute. The ventricles, responding to each second or each third of these stimuli from the auricles, beat for the most part regularly but at rates which suddenly vary from time to time. That is, the rate now suddenly doubles, now returns to normal, then trebles, and then, after a short time, returns to normal, etc. The pulse rate is seldom 150 per minute; usually it is nearer 100. This condition, while usually serious, may persist for months or for years and with few and slight symptoms.

Heart block, the more or less independent action of auricles and ventricles, is due to some disturbance of the auriculo-ventricular bundle (the bundle of His), which prevents all of the stimuli from the auricles from reaching the ventricles. Heart block sometimes is perfect and permanent; that is, this bundle remains permanently impassable for stimuli. The auricles in such a case beat with normal regularity and rate, but the ventricles, now receiving no stimuli from the auricles, create and control their own beats, contracting regularly about 28 to 30 times to the minute. In cases of **Stokes-Adams disease** the block is complete while it lasts but is temporary. In these cases the block develops suddenly, the heart for a moment stops entirely, and because of the resulting cerebral anæmia the patient faints and, if the pause is for over fifteen seconds, has a convulsion; then the ventricles begin their independent rhythm, and the patient recovers consciousness. Later, the normal rhythm again returns. In most cases heart block is partial, that is, all the time some beats get through, while others do not, and the result may be a regular pulse, as when each second stimulus gets through (the 2-1 rhythm); but as a rule the result is a very irregular pulse. This is best

shown by the electrocardiograph records, on which one can see that not all of the auricular beats are followed by those of the ventricle. Slight true heart block may occur in any acute fever, especially diphtheria, acute rheumatic fever, typhoid fever; in valvular heart disease; but often the cause is a gumma or other local lesion in the heart septum, which presses on or cuts the auriculo-ventricular bundle.

By endocarditis is meant inflammation of the endocardium, the membrane which lines the cavities inside the heart. This

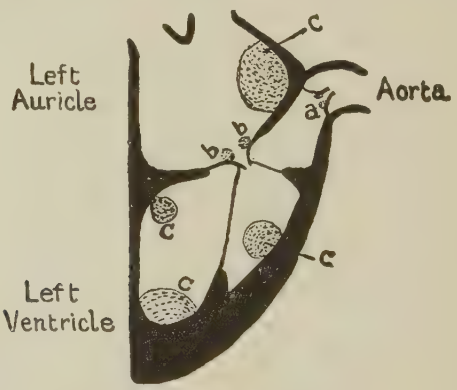


FIG. 43.—The vegetations of acute endocarditis in their most common locations. *a*, vegetations on the aortic valve (ventricular surface of flaps); *b*, on the mitral valve (auricular surface of flaps); *c*, mural thrombi.

membrane is practically the same as the intima of the blood vessels. Inflammation of the endocardium leads to the deposition, on the inflamed spots, of fibrin clots (thrombi), here called **vegetations,** because these thrombi project from the endocardium and in shape somewhat resemble the cauliflower.

Any part of the endocardium may be affected. A clot which forms on the wall of one of the cavities of the heart is called a **mural thrombus** (Fig. 43, *c*). Such clots, are, as a rule, rather large—about the size of a marble. They usually are several in number and when numerous may almost fill an auricle or the tip of a ventricle. These clots make very little trouble for the patient as long as they remain attached, but when one breaks loose it is swept along as an embolus

in the blood current into the arteries, and the result will depend on the vessel which it finally plugs.

But, unfortunately, endocarditis attacks oftenest the valve flaps themselves, and that part of these important structures where it can do the most harm,—that is, their margins, which are pressed together when the valve is closed (Fig. 43, *a*, *b*). The germs are deposited on the edges of the flaps, probably because the tissue there is subject to the greatest strain and is therefore most vulnerable. A fringe of these small vegetations along the margins of a valve flap is the chief cause of valvular heart disease.

There are several kinds of **acute endocarditis,** e.g. the simple, the subacute, and the ulcerative. This difference is very important. In **simple,** or **rheumatic, endocarditis,** the cause of which we do not yet know, the vegetations are like tiny translucent beads, about the size of the head of a pin. These tiny beads look harmless enough, but their late effects are often very important. Sometimes, perhaps, this valve never suffers any further consequence, but the rule seems to be that this simple endocarditis sets up a slow, insidious thickening of the flap, a process which after years makes it a little shorter, just a little thicker, than normal, and just a little shrivelled along its edges—not much but enough to prevent that flap from perfectly closing the orifice of the valve, and hence to allow a leak. These cases have practically no symptoms. Indeed one may not be sure that the valve has suffered any permanent injury until months or years have passed, and then the condition may be discovered accidentally. A patient with a slight leak in the valve can, by leading a quiet life, live years—perhaps an average lifetime—without suffering any especial symptoms. He may, indeed, never discover that he has any heart trouble until examined for life insurance or the army. But some unusual strain or severe exertion may overtax the heart, and death be the first sign of disease. In many cases a sudden strain is followed by months or years of invalidism.

The subacute bacterial endocarditis is due to Streptococ-

cus viridans. This causes a long, slow fever, lasting for months, which sometimes resembles typhoid fever, sometimes malaria, sometimes abscesses of internal organs, sometimes acute rheumatism, and sometimes all of these in succession. Patients with this infection are ill, but unless the heart is examined the nature of the disease may be entirely overlooked and the patients for weeks or months be treated for any one of many conditions. Its course usually ends fatally.

In malignant, or ulcerative, endocarditis the clots are larger and often hang from the valve flaps like stalactites, or are loosely attached to the valve by slender pedicles. They often break loose and travel as emboli. Sometimes many break loose, each one plugging some blood vessel, and then it seems as if the heart were bombarding the body. But these emboli may do more than merely plug a vessel, for they are sometimes full of bacteria, and, if so, where they settle an abscess forms. The spot on the valve flap where the clot forms is so much weakened by the inflammation that its edge becomes torn, frayed, and weak, with chordæ tendinæ broken or torn loose. In fact only tags and tatters of a valve may be left. The clots may calcify and feel like masses of shell. Between these two extremes (a slightly thickened flap and one totally destroyed) are flaps with all degrees of injury. This severe, malignant endocarditis seems to attack especially those valves which are the seat of simple endocarditis of longer standing. The ulcerative form causes very serious illness with high fever, sometimes severe chills, and the constant fear of emboli loosening and causing their damage. By blood culture we often can find the germ of ulcerative endocarditis.

We have spoken only of valve lesions which cause a leak, or a valvular insufficiency. There is another possibility. The inflamed flaps may grow together along their margins, or by contracting may narrow the orifice of the valves, or may become very stiff and protrude into the orifice. The result

is called stenosis of a valve. Such valve orifices, although smaller than normal, usually leak also.

As far as we know, acute endocarditis is always due to some germ. The germ of simple endocarditis has never been identified, but this disease often follows acute rheumatic fever, and many think that a germ which causes both the joint and heart inflammations enters the body through inflamed tonsils. St. Vitus's dance also, often associated with acute rheumatism, is in many cases followed by acute simple endocarditis. The cause of this, however, we do not know. Subacute endocarditis, on the other hand, is due to Streptococcus viridans, and the ulcerative forms are due to several germs which we know. Scarlet fever, erysipelas, diphtheria and pneumonia are frequently followed by serious heart disease. The germs of these diseases are carried to the heart by the blood, and the heart valves are favorite places for these germs to settle.

In very young children the valves of the right heart are more often affected; in older children and adults those of the left. Of all valves the mitral is most often affected, the aortic next oftenest.

The treatment of endocarditis seems hopeless, but it is not. Our first aim should be to remove every infection which might have started the trouble. It is true that by doing this we do not stop the trouble already present, but endocarditis, like many other chronic infections, is a series of infections, and by removing the source we stop the repetition of infections. Then, the patient should rest perfectly quietly in bed while the carditis is acute, until the temperature is normal and for a long time afterwards, up to a time when the muscle is strong and has had plenty of time to become used to the new conditions, that is, to the leak with which it must work. This may mean rest in bed for months after the patient feels well. During this time an ice bag is kept over the heart, a few blisters are raised at the apex, and suitable medicines are given.

Chronic endocarditis is the result of the acute endocar-

ditis; it is the resulting permanent injury to the heart. But there is another form of chronic endocarditis, which affects especially the aortic valves, which is very important. We mean the **arteriosclerotic form**; for the same process which causes thickening of the intima of the aorta can creep over to the aortic valves—which are really but folds in the intima, called here the endocardium—and produce slight thickening, stiffness of the flaps, and slight curling of their edges—not much, but just enough to prevent a perfect closure of the aortic orifice.

Angina pectoris is the name given to the sudden agonizing pains in the region of the heart, which run down the left arm sometimes to the little finger and ring finger of the hand, which patients with arteriosclerosis of the coronary arteries and of the root of the aorta suffer. In this pain the patient holds himself immovable, breaks out into a sweat, and fears impending death. These attacks may come on without apparent cause but often follow an exertion, mental excitement, or exposure to cold.

The **treatment** of true angina pectoris is the treatment of the arteriosclerosis, especially if this be luetic. For the pain the immediate inhalation of amyl nitrite is best. One proper dose of this drug is sealed in a small glass tube (called a "perle"), which, when need arises, is broken inside a handkerchief or towel and at once held under the patient's nose that he may inhale it. Morphia in large doses hypodermatically, or even chloroform may be necessary. For the less severe attacks one tablet of 1/100 of a grain of nitroglycerine, put immediately on the tongue, will give some relief. Many patients always carry these tablets in their pockets. Recently the operation which removes the superior cervical sympathetic ganglion of the left side of the neck has been performed with great success in the treatment of severe angina pectoris.

In addition to true angina pectoris many patients have heart pains, or, better, **precordial pains,** since the patient locates these pains in the chest wall over the heart. These, like the pains of true angina, are accompanied by soreness

in the skin of this region, for each internal organ is represented by an area of skin which "suffers with it." Fortunately, treatment of this "area of representation," as it is called, relieves to a limited degree the trouble with the internal organ which it represents. This is the justification of our application to the skin of mustard plasters, hot-water bottles, electric pads, leeches, and blisters—treatments which may not be very important, but which do have some value, if only for comfort's sake. These precordial pains are very hard to interpret, because so many of them are nothing but neurasthenic symptoms due to the apprehensions of a person tormented by the fear that he has heart disease. Others mean some real heart weakness, and these come especially after exertion.

Coronary thrombosis. We should always be on the lookout, when a patient has such pains as we have described, especially the substernal precordial pains, for **coronary thrombosis,** that is, for closure of a coronary artery, with resulting infarction of the heart wall. This is the cause of most of the sudden attacks of heart failure and of many of the fatal attacks of angina pectoris. In cases of coronary thrombosis the wall of the artery before the attack is definitely sclerotic. Then a thrombus forms. The patient has a sudden anginal pain or a series of such pains, or, more often, of dull substernal pains, with more or less shock. Then, in a few hours develop fever, leucocytosis, and, later, a friction rub over the pericardium. Often the pulse is irregular and the blood pressure low, and the patient from the first feels more or less ill. The possibility of recovery will depend on the size of the infarct. Small infarcts heal, larger ones do not, and after a few days the patient suddenly dies. Those who recover have later the danger of an embolus, since the infarcted area of the heart wall will be covered on its inner surface by a blood clot which may, but which need not, give rise to emboli. These patients should be kept in bed and very quiet; they should not be allowed even to turn themselves. They are given a low diet, and morphia to prevent restlessness.

Valvular Lesions

The strength of the heart resides in its myocardium. Apart from the danger of emboli, the danger in endocarditis is the myocarditis which always goes with it, for an infection cannot cause the former without causing more or less of the latter also. If the myocardium be normal the heart can function with little evidence of weakness even though a valve is badly injured. We know an active business man, seventy-two years old, whose mitral valve certainly is in very poor condition. He likes to tell us that when twenty years old he had heart disease and "was given less than two years to live"; and yet for fifty years "he has had good health." Such cases are not very rare. What happened in this case was that the source of the acute endocarditis disappeared, and that therefore the endocarditis subsided, leaving the valve permanently injured, but the myocardium in good condition, thus giving him a good enough heart. Most cases are not so fortunate as this, for, unless properly treated, the infections which cause the endocarditis and the myocarditis usually continue and flare up frequently, each infection adding its little injury to the troubles already there.

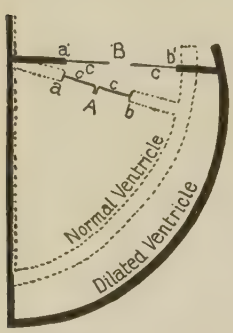


Fig. 44.—A diagram of a normal and a dilated ventricle. When because of weakness the muscle wall stretches, and the cavity of the ventricle "dilates," the orifices will dilate also. The valve flaps *c*, can close tightly the orifice *a-b*, but cannot close the orifice *a'-b'*, and yet there has been no change in the flaps *c*. The valve *A* is "competent," but *B* is relatively "incompetent," or "insufficient" (-ly closed).

If a valve **leaks**, the condition is called valvular **insufficiency** or valvular **incompetence.** When the orifice is too small, **stenosis** is said to exist. If the valve flaps are so torn or shrunken or stiffened that they cannot fill a valve orifice of the normal size, the condition is **absolute insufficiency. Relative insufficiency** is a different condition. The heart is an elastic, hollow, muscle bag (Fig. 44), and its capacity is dependent on the "tone" of its walls. When extra strain

occurs and the walls are unable to hold their tone, they "stretch," and the volume of their chambers is increased. But the size of the orifices in these chambers will increase also. The flaps, *c*, of a normal valve are of just sufficient size to fill an orifice of normal size, and they cannot stretch. So that, when the orifice is larger than normal, the normal flaps cannot close it; that is, the valve is relatively insufficient.

The normal heart uses, under ordinary circumstances, only **a small part of its strength** for each beat; the rest is held in reserve. But let a normal person run violently or lift some heavy weight, and the heart must fall back on its reserve strength. If the total strength of the heart is represented by the line *a-b* (Fig. 45 *A*), under ordinary circumstances *b-c* will represent the force necessary for each beat, and *a-c*, the force held in reserve, which it can use when subjected to extra strain. A runner is said not to run a successful race unless he faints just when he crosses the tape; that is, unless at that moment his heart has reached the limit of its available reserve strength. (There is normally a reserve beyond this.)

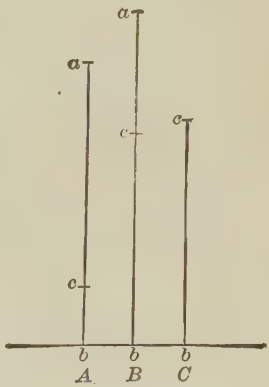


FIG. 45.—Diagram of cardiac force. *A*, normal heart. *B*, heart hypertrophied because of a leaking. *C*, heart with "broken compensation." *a-b*, total heart force; *a-c*, reserve heart force; *c-b*, force used in each contraction.

The heart valves, while very important for normal heart action, are, nevertheless, not indispensable, for the entire circulatory apparatus (which includes not only the heart but the arteries, veins, and capillaries, as well as the organs of respiration and, certainly, the great cardiovascular regulating nervous system) can function efficiently even though one valve is badly injured, provided the heart muscle remain strong. All the changes in the functions of the circulatory apparatus which follow a valve lesion—changes in the method of heart beat, hypertrophy of the myocardium,

changes in blood flow—are called **compensatory changes,** since they develop in order to make up for, i.e. eliminate, the evil effects of a valve defect. These compensatory changes will be mentioned later with the description of the various heart diseases.

Aortic stenosis. Of the various valvular lesions we shall describe the narrowing of the aortic valve, a rather rare lesion, first, because it presents the simplest mechanical problems. In this disease the flaps of the aortic valve have become glued together or have become so stiffened by masses of exudate or even by calcareous plates that they project into the lumen and partially occlude it (Fig. 46, *B*). The orifice of the aortic valve, which connects the left ventricle with the aorta, is therefore smaller than normal, and naturally the

FIG. 46.—The aortic valve. *A*, normal valve. *B*, the flaps of the valve are so thickened by vegetations that the orifice of the aorta is partially occluded, "aortic stenosis." *C*, the valve flaps are so torn that they cannot close the orifice, "aortic insufficiency."

left ventricle must work harder than usual when it tries to force the usual amount of blood into the aorta. In order to prevent any failure of circulation, the ventricle therefore must contract more slowly but with greater force than normal (a part of the compensation). The first sound will be long-drawn, rough, and vibrating in character—"r-r-r-r-r-rub-dub." This very characteristic rough systolic murmur, though it is sometimes the most musical of the cardiac murmurs, may be the harshest and loudest of them all. It is loudest at the aortic area and is transmitted into the great vessels.

If one puts his hand over the base of the heart, especially over the aortic area, of such a case, he feels a vibration which is the most intense of all cardiac thrills, and which resembles the purring of a cat. This is caused by the forcible squeezing of the blood through a small hole.

The pulse will be rather slow, for the heart must make slow, laborious contractions. The ascent of the wave is gradual, because the blood is squeezed slowly into the aorta, which explains why the apex of each wave is rounded (Fig. 47); but the blood must move fast, since an amount as nearly normal as possible must flow in each minute.

In this case the left ventricle only has a harder task than usual to perform. Its walls will be stretched a little at first and it will use more of its reserve force, but gradually it will tend to do what all muscles do when given increased work—it will become stronger and thicker; that is, it will **hypertrophy.** This condition may be represented by the **line** *B*, of Fig. 45. The total strength of the ventricle *a-b*, is greater than normal; but since its task at each beat, *b-c*, is harder, the margin, *a-c*, is much less. The man easily finds this out when he undertakes any unusual exertion.

FIG. 47.—Pulsus tardus of aortic stenosis.

In the course of time the heart, if its muscle is fairly normal, will gain enough extra strength to enable it to fulfill its functions, that is, to pump each minute the required amount of blood into the aorta. Then, we say, the lesion is compensated, the heart is in a **state of compensation.**

In aortic stenosis the trouble is limited to the aortic valve, and the extra strain affects only the left ventricle. The rest of the heart theoretically remains undisturbed, and the circulation progresses without any change. Now, suppose the lesion at the aortic valve becomes extreme, or suppose that, because of some unwise strenuous effort on the part of the patient, the heart becomes so "strained" that it has no reserve left. Then the left ventricle cannot throw out its hundred cubic centimeters of blood at each beat, and a "break in compensation" results; that is, *a-c*, has disappeared, and most of the available energy is used to accomplish each beat (Fig. 45, *C*). The left ventricle is filled each time by the

auricle with the usual amount of blood, which it can with difficulty expel. Of course it becomes overdistended; that is, the heart—a hollow muscle bag—becomes stretched, the orifices into it become larger, and soon the mitral-valve flaps, although normal, are unable to close the unusually large mitral orifice. Then all the results of a mitral insufficiency will follow. During the state of compensation the patient may have had no hint that his heart was at all abnormal. He may have noticed that he was not quite so good a runner as formerly, but that may have been all. When the heart reaches the state of non-compensation, however, all the symptoms of dilated heart will follow, and these are severe enough.

We have used aortic stenosis as the first example of a valve lesion because its mechanical problems are so simple, but, as a matter of fact, aortic stenosis is a disease especially of elderly men with marked arteriosclerosis, rigid chest, and emphysematous lungs, and so compensation usually is very poor.

Aortic insufficiency. When the aortic valves cannot quite close the aortic orifices (Fig. 46, C), then the blood, always under high pressure in the aorta, will hiss back into the left ventricle during diastole; that is, between beats, while the heart is relaxed. That is, a condition of aortic insufficiency exists. One must remember that the aorta and its branches, the arteries, form a very elastic tree kept overfilled by the heartbeats. The elasticity of these overdistended arteries is always forcing the blood on into the smaller vessels, but, if not prevented by the aortic valve, will force it also backwards into the heart. If all the blood forced out by one systole should leak back during diastole, circulation would stop. The heart overcomes this difficulty much as a man climbs a slippery hill. If for every upward step of two feet in length he should slip back two feet, he would make no progress; he will try to take steps perhaps three feet long, in order that each step may leave him at least one foot higher. So the heart will try to throw out into the aorta so much blood that the normal amount will in spite of the back flow

remain in the aorta. The body helps to meet the problem by relaxing the peripheral blood vessels so that they can quickly receive the blood thrown into the aorta by each heartbeat, as a result of which we find the diastolic pressure to be low. It is this rapid flow onward which explains the Corrigan pulse, not the back flow. The ventricle at each contraction throws this large amount of blood into the aorta with such force that the wave can be seen even in the smallest arteries, while the large arteries seem really to jump. As might be expected, the pulse wave when charted has a sudden high ascent, a sharp apex, and a quick fall (Fig. 48).

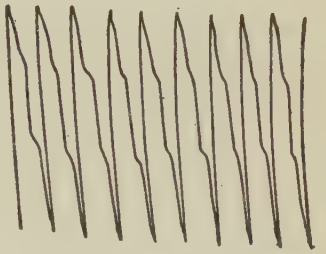


Fig. 48.—Pulsus celer of aortic insufficiency. Corrigan's pulse.

Because of this extra amount of blood to be thrown out by each beat the left ventricle dilates, and, since it takes more muscular effort to accomplish each beat, the left ventricle hypertrophies, and to such a degree that hearts with this lesion are the largest hearts seen in any disease. If the dilation is not extreme enough to stretch the mitral orifice, the increased strength of the left ventricle, together with the peripheral vascular dilatation, will compensate for the whole trouble, and the circulation will continue normal. A patient with aortic insufficiency may for years be unconscious that he has any heart trouble at all; or he may complain of palpitation on exertion, perhaps of dizziness on sudden movements; or he may discover the trouble only by accident. On examination, however, his heart will be found larger than normal, the hiss of the backward flow may be heard (Fig. 49), and the character of the pulse will be quite unmistakable.

There are **two groups of cases of aortic insufficiency.** The first is **the endocarditic group,** in which the trouble is due to rheumatism. Here an acute endocarditis has injured the flaps. Such a heart is able to compensate well even for an extreme lesion. The second is **the arteriosclerotic group.**

This is a different matter. The flaps of the valves are really folds of intima, and the arteriosclerotic changes of the aorta will spread to, thicken, and shorten, the valve flaps. But compensation here is not easy, for the coronary arteries, which provide the heart muscle with blood, open from the aorta behind the aorta valve, and they also are injured by the arteriosclerosis, which narrows their mouths and creeps along their course. In order to hypertrophy, the muscle must have increased food, but in this case the source of supply is impaired by the disease of the coronary arteries, and so the arteriosclerosis not only causes the valvular lesion but hinders the hypertrophy necessary to compensate for it.

Patients with aortic insufficiency usually have a characteristic **pallor** described as "earthy color," not as a rule due to anæmia, and **visibly jumping superficial arteries,** each of

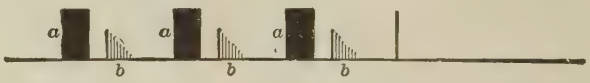


FIG. 49.—A graphic representation of the heart sounds in a case of aortic insufficiency. The second sound *b*, is replaced by a "diastolic blow." (Compare with Fig. 39.)

which often suggests the diagnosis. The **Corrigan pulse** may be so pronounced that a diagnosis can be made when one is shaking hands with the patient. The pulsation of the abdominal aorta may shake the patient's whole body and the bed on which he lies. If the trouble began early in life the whole precordium may bulge and pulsate. The pulsation may heave the chest wall beyond the cardiac limits, as far laterally as the anterior axillary line. The enlargement of the heart is such that the apex is displaced a little to the left and **to a greater degree downwards,** so that the apex beat usually is in the sixth or seventh left interspace, in, or just beyond, the nipple line.

Mitral insufficiency. The mitral valve is often attacked by endocarditis, and its common lesion is mitral insufficiency. Let us suppose that a little curling, shortening, fraying, tearing, or stiffening of one or both of the mitral-valve flaps (Fig. 50, B) prevents a perfect closure of the valve orifice during systole—that is, while the powerful ventricle is forcing the

blood into the already overfilled aorta. Then the ventricle will force some blood back into the auricle. During the systole the left auricle should receive blood from the pulmonary veins alone, blood which it will pass on to the ventricle at its next beat. But now during systole the auricle gets blood from two sources, from the lungs and from the ventricle, and from the latter under high pressure. It must, therefore, dilate, and, since it has extra work to do, it must hypertrophy. But this back-flow from the ventricle also checks the current of blood from the lungs, damming it back upon the right ventricle, which must keep up the pulmonary circulation against this, a new obstacle. The result is that even a slight mitral leak will lead to dilatation and hypertrophy of the right ventricle,

FIG. 50.—The mitral valve. *A*, normal valve. *B*, the valve flaps are shrunken and torn so that they cannot close the orifice,—"mitral insufficiency." *C*, the valve flaps are so thickened that the orifice is partially occluded. The small circle represents the maximum orifice in some extreme cases,—"mitral stenosis."

and the pulmonary blood vessels will be constantly overfilled with blood, which explains the patient's frequent attacks of bronchitis. It is evident that in the presence of a mitral lesion perfect compensation limited to changes in one chamber of the heart is impossible.

A patient with a well compensated mitral insufficiency which began in childhood often is small in stature, rather puerile in appearance,and has fingers with bulbous ends suggesting drum sticks. His skin in general and the mucous membrane of his lips and cheeks are more or less visibly suffused (wine-colored), while the skin of the ears and the extremities may be definitely cyanotic. After exertion his skin surface in general may become rather deeply cyanotic. With passing years the venules of the cheeks become permanently dilated. Even while compensation is good (i.e., for a mitral

lesion) shortness of breath on exertion, palpitation of the heart, and cough due to the chronic passive pulmonary congestion (see page 120) are common symptoms. The pulse may be regular, of good volume, and of rather low pressure, but often, especially after exercise, it is somewhat irregular because of the presence of extrasystoles and perhaps also of a slight fibrillation, which may persist indefinitely, and which is not inconsistent with good compensation for a mitral lesion.

The cardinal signs of mitral insufficiency are a transverse enlargement of the heart to right and left; a systolic murmur, of maximum intensity at the apex, which is transmitted towards the axilla; and an accentuated pulmonic second sound. The precordium may visibly bulge and pulsate with the wide spread and forcible systolic impulses indicative of hypertrophy and dilatation. The apex beat is displaced horizontally

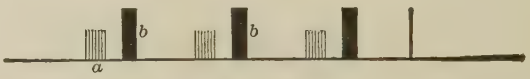


FIG. 51.—A graphic representation of the heart sounds in a case of mitral insufficiency. The first sound is a "systolic blow." (Compare with Fig. 39.)

to the left, beyond the nipple line and even to the anterior axillary line. The right ventricle may pulsate markedly and even produce a visible tumor below the left costal border.

Mitral Stenosis is a very common trouble, especially in women. In this disease the endocarditis of the mitral-valve flaps has resulted in an abnormally narrow mitral-valve orifice (Fig. 50 ,*C*). Normally one should be able to put three fingers through this orifice with ease, but in some well marked cases of stenosis one can hardly force a lead pencil through it. The valve flaps have grown together; the chordæ tendineæ have become much shortened, pulling the flap edges almost down to the tips of the papillary muscles. Or, masses of calcified thrombi make the stiff flaps project well across the orifice.

In mitral stenosis the left ventricle is not affected and can pump with ease all the blood it gets; but the left auricle has great difficulty in forcing its blood through this narrow orifice, and therefore it dilates and hypertrophies. The strain, there-

fore, falls back on the right ventricle, which now must "push from behind," so to speak; that is, it must so increase the pressure in the pulmonary vessels that this will help force the blood through the narrow mitral orifice.

Mitral stenosis, especially that which has developed during childhood, may produce a conspicuous pulsating tumor, due to the hypertrophied right ventricle, which occupies the region of the lower sternum and fifth and sixth left inter-spaces. The pulse of patients with mitral stenosis is small in volume and often irregular. The apex beat, which is in about the normal position, is not easily located by inspection, but on palpation it is discovered to be the most forcible, the most sharply limited, and **most characteristic of all cardiac pulsations.** The hand may feel as if struck by a hammer. At this point also, over an area not more than an inch in diameter, is usually felt and heard a **characteristic thrill** due to the auricular systole. It is purring in character, crescendo in intensity, and leads up to, and ends with, the shock of the first sound. It is more intense during expiration. Sometimes it is present only after exercise. It is always more pronounced when the patient lies on his left side. In a well marked case one gets, on palpation as well as on auscultation, the hint of a slight pause or hesitation which seems to separate the end of the thrill from the beginning of the shock of the first sound, and which may make the latter seem more forcible than it really is. This pause is explained by the delay in the transmission of the stimulus over the auriclo-ventricular bundle. The shock of the second sound usually feels much like a tapping at the pulmonic area. The presystolic rumble may be absent, but, if so, the **characteristically abrupt, slapping first sound** will be almost diagnostic. On the other hand, in spite of the spectacular character of the physical signs of this disease there is no valve lesion in which the heart sounds are as often perfectly clear. It takes strength to produce the signs mentioned above, but when the heart is weak the sounds may be perfectly clear, almost normal, yet with little differences in accent and rhythm which indicate much more danger than the loud

rumbles, thrills, and shocks which will develop when the heart becomes stronger. In some cases one hears at the pulmonic area a soft diastolic blow, the **Graham Steele murmur** of relative pulmonary insufficiency. This diastolic blow would seem to be characteristic of an early mitral stenosis. It is therefore heard most often in children. It is heard over the body of the heart.

Tricuspid insufficiency, due to disease of the tricuspid valve, occurs in children but rarely in adults; but relative tricuspid insufficiency occurs in practically every case of badly dilated heart.

The symptoms of **tricuspid insufficiency** are marked. At each beat the right ventricle forces blood in two directions—through the pulmonary valve, the normal direction, and back through the leaking tricuspid valve into the right auricle. This stream of blood meets the streams from the venæ cavæ and hence impedes the whole venous flow towards the heart. The veins are everywhere overfilled with blood, which is at first dammed back and later impounded. The patient is blue (cyanotic), especially as to his lips and fingers. **A pulse** similar to that sent by the left ventricle through the aorta to the arteries is in such a case sent by the right ventricle **to the veins. The liver,** now swollen to perhaps two or three times its normal size, **pulsates** because of this venous pulse wave. The walls of the stomach and bowels, of the kidneys, and of all other organs, are turgid with venous blood and do not receive their proper supply of fresh blood. Since this prevents the internal organs from doing their work well, we get symptoms from each of them. This condition is called "chronic passive congestion" of the stomach, liver, kidneys, etc.—chronic because of its duration; passive because the congestion is the result of blood dammed back into the organ because of trouble ahead and is not caused by any trouble in that organ itself. If the stomach (or kidney, etc.) were inflamed because of local disease, its vessels would be congested. That would be an acute "active" congestion; this is passive. The skin of the legs and dependent portions of the

body become œdematous, i.e. dropsical, or water-logged. Serum collects in the abdominal cavity (ascites) and in the pleural cavities (hydrothorax, or "water on the chest"). If in any way we can make the heart "competent," circulation will at once improve, the congestion of blood in the various organs will be relieved, and all symptoms due to it will disappear. Chronic passive congestion, however, is not always the result of tricuspid insufficiency. It develops in the lungs of patients with even slight and perfectly compensated cases of mitral insufficiency and in the abdominal organs of many patients with slight cases of cardiac diseases. In these cases the congestion seems to have a protective function, since it decreases the total volume of blood which the crippled heart must pump around the body.

Pulmonary valve disease occurs, but it is very rare except in children.

It should be emphasized that **not all heart murmurs mean heart disease**; in fact, many of them are produced in the lung near the heart. The heart becomes smaller during each beat, and the potential vacuum thus created is filled by the neighboring lungs, which expand, that is, which inhale; and this produces a respiratory blow. It is also true that very weak hearts may produce very clear sounds and later, when they become stronger, produce loud murmurs. Indeed, **the sounds of the heart are a very poor criterion of the heart's health.** The changes in its size, its regularity, the pulse, the blood pressure, and especially the heart's ability to meet an extra demand—these are much more valuable.

Many fairly normal persons with symptoms suggesting heart trouble are sure they have a serious heart disease; and many with serious heart disease have no symptoms suggesting heart trouble. The patients with real cardiac troubles suffer mostly from weakness, shortness of breath, cough, swelling of the feet, and disturbance of the liver, stomach, and bowels. They are seldom conscious of their heart action, no matter how irregular or tumultuous it may be. Even those with heart blows so loud that they can be heard at a distance

look curiously about the room to see where the sound comes from. Those who feel the heart pound (palpitation) or "skip" or "race" are usually those nervous patients with fatigue neuroses or those who smoke too much, or who drink too much coffee; and such persons are seldom very ill.

Treatment and Nursing Care

In the treatment of cardiac cases we aim first, to relieve any inflammatory conditions in the heart itself (endocarditis) and also in the lungs; to keep the pulse rate as slow and regular as possible; the blood pressure, within as normal limits as possible; the respiratory movements as unrestricted as possible; and finally to continue this treatment for a long time.

Tonsils should if infected be removed early, the nasal sinuses drained, and any bronchitis treated, otherwise our patient, finally in satisfactory condition, suffers a reinfection from these foci and we have the whole treatment to repeat. In many cases the cardiac decompensation itself is the direct result not of heart strain but of the flaring up of a chronic infection and compensation requires merely that this, e.g. an attack of bronchitis, improve.

In the treatment of all cardiac cases **rest in bed is the first essential.** Especially if there is fever, suggesting an endocarditis or bronchitis, etc., the patient should lie as horizontal as possible, since in that posture the pulse is slowest and blood pressure is lowest. A patient with dyspnœa however, and especially if he has orthopnœa, cannot do this and should be made comfortable in that position which gives him the greatest ease. The Gatch bed is the most comfortable one for patients with cardiac dyspnœa. The mattress should give the head, back, shoulders, and arms firm support, and the knee hinge should prevent the patient from slipping down. Every detail of posture should have as its aim the freest expansion of the lungs, in order that the vital capacity of respiration be as great as possible. Therefore the pillows should not push the shoulders forward, or allow the head to fall forward on the chest. The weight of ice-bags, poultices, and bed-clothes should be minimized. If a patient with orthopnœa desires to

lean forward he should have a proper support, or be made comfortable sitting up in an armchair with arms conveniently shaped as a support for his elbows so that he can by leaning on them raise his shoulders. In this posture the extremities may suffer but there will be better circulation in the lungs and brain. If dropsy is present the patient should be in bed with the extremities elevated. Massage and firm bandages may improve the circulation of œdematous limbs. If the chest contains much fluid, this should at once be removed by aspiration in order to relieve the organs of respiration. If there is cyanosis and much congestion of the viscera, a prompt venesection is indicated.

It is important, to remember that a continued stay in bed is not necessarily a rest. Mental worry, apprehension, and any physical discomfort due to the bed, to sores on the skin, or to the temperature, light, or humidity of the room may defeat the purpose of the physical rest.

When treatments are given, the bedpan used, or the meals served, the patient's comfort should be a prime consideration. He should not be allowed suddenly to sit up, and all possible physical sources of exertion and strain should be avoided. An ice-bag applied to the anterior chest wall to relieve pain or to stimulate the heart muscle should contain as little ice as will accomplish the desired effect, all air should be expelled, the water from melted ice frequently emptied, and its weight supported if possible by a cradle.

Comfort is essential and every known art should be used to insure it. The room should be bright, cheerful and quiet, the surroundings congenial, free from disorder or confusion. Fresh air is essential. This is not a problem of increasing the amount of oxygen, for of that there will always be a surplus, but is one which concerns the humidity and temperature of the rooms. A slight change in these may increase the patient's discomfort and difficulty in breathing to a distressing degree.

Worry, anxiety, and irritation, in fact all causes of mental or nervous strain, should in so far as is possible be avoided.

To accomplish this the **patient must be physically as comfortable as we can make him**; the entire conduct of his treatment must be so manifestly efficient as to inspire his confidence; no choices should be left for his decision. His visitors should be limited to a few, they well chosen, and their visits brief, for the breathless patient should not be forced to talk or even to listen to conversation. To get mental serenity we may have to use drugs, and we do not hesitate to do so, using morphia in small regular doses, one tenth of a grain every four hours, to quiet the heart, both by quieting the patient's mind and also by decreasing the number of reflex stimulations which the heart receives through the nervous system.

Prolonged sleep is essential, and during the day also the patient should be kept in as drowsy a condition as possible in order to eliminate, as far as we can, all of those emotional episodes which stimulate the heart. The character of the sleep also is important, since unpleasant dreams can cause serious changes in the blood pressure and heart rate. To prevent these sedatives are frequently necessary.

Daily cleansing baths and frequent massage refresh the patient, stimulate the circulation, and keep the skin in good condition. Chafing and irritation are to be particularly avoided in cases with œdema. The patient should be frequently turned and all pressure points protected to avoid bedsores.

Diet. The problem connected with the diet of cardiac cases is of major importance. The essential point of a cardiac diet is that, while sufficiently nourishing, it favors a low position of the diaphragm and its free play during ins- and expiration, for even a slight elevation of this causes great discomfort. Because of the chronic passive congestion of the digestive tract the appetite may be poor and digestion disturbed. **To improve the appetite** the mouth should be kept clean and the food given in frequent small meals at very regular intervals. In general, therefore, a **cardiac diet is one which keeps the stomach as empty as possible.** Such a diet is light, dry, and not rich in cane sugar. That is, one should have in mind the problems created by solids, fluids

and gases. We usually prescribe eight small meals a day, of concentrated food, and at regular intervals. The total food value for the day should be as little as it may wisely be, since in general we would like the patient to lose weight, and its volume in the stomach at any time should be as small as possible. To illustrate: a five-dollar gold piece has exactly the same money value as have five silver dollars or five hundred coppers, but the five-dollar gold piece is much easier to carry around than are its equivalents. So one egg equals in **total food** value a half glass of milk or two average servings of spinach. These three contain the same amount of food, but how much they differ in volume! The cardiac diet, therefore, consists of milk, eggs, scraped beef, butter, fruit juices, and other concentrated foods. We assiduously **avoid those foods rich in cellulose,** therefore, whole wheat, cereals, and the coarse vegetables. The patient if troubled with difficult breathing should be instructed to take his solid food in **small mouthfuls,** and to chew the food thoroughly, unless chewing is too great an effort, in which case liquids should be employed. **The meals should be accurately timed** since regularity creates good visceral habits which aid greatly to minimize disturbances of the digestive functions. Foods should never be forced because, if the patient cannot be tempted, the chances are that the digestion itself is at fault. To maintain regularity, however, he is given a few mouthfuls at least at each proper time, and if this seems unwise then that meal is omitted but no more food offered until the time of the next regular meal.

It is very desirable that the patient **lose weight** (unless already emaciated) for each pound lost is a not inconsiderable mechanical relief to the circulation. To avoid that faintness or sleeplessness due to hunger a dry biscuit and a small glass of milk may be given regularly just before bedtime and again early in the morning.

Another way to keep the stomach almost empty is to **limit the amount of fluids.** Water is in general restricted because it tends to increase the œdema and because its trans-

portation to the kidneys certainly adds to the burden of the heart. The daily total volume of all fluids given should be measured and charted, and should not exceed 1500 c.c.s. This amount should be compared with the daily output of urine, so that we may recognize early a tendency to the retention of water which might indicate increasing cardiac insufficiency or impairment of the kidney function. Since caffeine is an excellent heart tonic, we allow coffee and thus avoid the bulk of that water which would be given with medicine; and, since tea is for some a good sedative, we allow this to those who desire it, but in neither case should cane sugar be used to sweeten the drink.

Again, we keep the stomach empty by **avoiding those foods which form gas easily** because a stomach inflated by gas disturbs the heart even more than one filled with solids or liquids, since the weight of the latter tend to pull the stomach down. The foods which easily produce gas in the stomach are the cooked (therefore quickly digested) starches, the pure sugars which easily form glucose, and foods containing a good deal of sulphur, like peas, beans, potatoes, and cabbage.

The bowels must be kept as nearly empty as possible of solids, fluids, and gases, since if inflated they press up against the stomach, and this against the diaphragm. To effect this, cathartics are used, especially compound licorice powder, jalap, aloes, calomel, senna, and phenolphthalein; but less often the salines.

Therapeutic baths when carefully given have some value, but baths much above or below the body temperature should never be given without a doctor's order. They have a marked effect on the peripheral circulation and therefore upon the heart. **Warm baths** may act as a sedative, but are dangerous unless well controlled. They should never be given in a very warm room; certainly the patient should never enter the hot room of the Turkish bath. **The Nauheim baths** (temperature below 92° F) are of benefit because of the effect of the ebullition of the carbon dioxide gas liberated against the

skin. Sea bathing is sometimes ordered to tone up the whole system. In this case the high specific gravity of the water has physical therapeutic value.

Of medicines, caffeine in any form is one of the best heart stimulants, whether given as coffee, or in pure form by mouth, or hypodermatically as caffeine sodium benzoate, in one-grain doses. **Digitalis** of course is the most important heart drug, but unfortunately is badly abused. It is used far too often, far too early in the case, far too long, and usually without any clear understanding of what we expect the drug to accomplish. Digitalis therapy should not begin until the patient is comfortable, the bowels well cleaned out, and the stomach quite relieved of any nausea which may have been there. This usually means not beginning before the second or third day after a patient with broken compensation is placed under observation. We may give digitalis as the powdered leaf or the tincture or the infusion. There is no particular advantage of one over the other, but probably the powdered leaf is the best. This is usually given four times a day in doses of at least 1½ grains. Digitalis should not be given unless the patient shows some definite physical sign which we are confident digitalis can relieve. Then, the dosage we use is not the quantity mentioned in any text book but the quantity necessary to abate in the patient that particular symptom which we have chosen as our signal. If the heart is fast and irregular we should give enough to slow it down. Of course we must be careful that we are not deceived by a bigeminal pulse. We do not give digitalis in small doses over nearly as long a period as formerly, but we now give it in large doses until we get the result we are looking for. This is true of all our drugs. Formerly several medicines were given three times a day for months. While we believed that there would be some benefit from such medication, there was no way in which we could be sure. Theoretically, the right dose of morphia in a case of renal colic is the quantity which will actually quiet the pain; the dose of castor oil is the amount which actually moves the bowel;

the dose of adrenalin in asthma is the amount which actually relieves the bronchial spasm. So the dose of digitalis is the amount which will slow down a rapid heart. Of course we must have a limit in all cases, and that of digitalis on the average is the equivalent of one cubic centimeter of the tincture for each ten pounds of weight of the individual. That is, a patient who weighs 150 lbs. is thoroughly "digitalized" when the body contains at one time 15 cm. of the tincture. Since the body destroys about 1.5 cm. each day, we must make allowance for this. For example, if we intend to digitalize the above mentioned patient in three days, we must give him 18 cm. during those three days, since within that time the body will have destroyed 3 cm. Since each 1 cm. of the tincture of digitalis is equal to 1½ grains of the powdered leaf, we can easily calculate how much of the latter to give. It seems to make little difference how rapidly we give digitalis. Of course not all persons respond alike to digitalis, hence the need of care in observing any sign of heart weakness.

In the early treatment of the case, while the patient is in great distress, we give various hypodermic preparations—especially camphor-like bodies (e.g. cardiazol), preparations of strophanthus (ouabain, 0.5 mg.), and, in great emergency, adrenalin.

The criticism is made that we keep patients with the lesser grade of cardiac decompensation too much or too long in bed, and much too quiet, also, that if prolonged stay in bed is necessary, we do not re-educate them back to more activity carefully and deliberately enough. Undoubtedly several weeks or months in bed, even though it may, as in the fever cases, be the lesser of two evils, is a treatment not without its dangers, and in the milder cases there are definite advantages in allowing the patient some mild exercises.

Exercise. When the reserve cardiac force is restored, the patient is **gradually allowed more and more exercise.** This must be carefully planned. Too often this exercise is defined in terms of sitting up, standing up, and walking, all

very violent forms of exertion since the patient must handle the entire weight of his trunk. Much better, the patient, lying flat on his back or sitting up in bed, repeats certain systematic free arm and free leg movements. Later, he sits up and much later he walks increasing distances. The exact length of his walks and the grade of the paths followed should be accurately measured. Also, the speed of the movements is of many times greater importance than is mere distance. Exercise which gives pleasure without discomfort is best. Exercise in the open air is to be preferred. It is important to remember that exercise enjoyed with comfort on one day may on another day because of changes in temperature, humidity, and wind be quite impossible. Also that worry, want of sleep, or gastric disturbances, may interfere with exercise previously possible.

The Schott exercises are of value if carefully supervised. These are arm movements made against the regulated resistance of a nurse or trainer. In any case the exercises followed should be systematically well planned, otherwise the care of weeks may be suddenly lost through some indiscretion. The doctor should prescribe the amount and form of exercise and the nurse should watch its effect on the patient, noting both the subjective fatigue produced and the time required before the quickened pulse returns to its previous rate.

Before discharging a patient, no matter how great his improvement, unless already done the tonsils, if infected, should be removed, abscessed teeth pulled and nasal sinuses drained. In this way we may avoid repetition of the attack of decompensation. The patient should be taught to move always deliberately; to eat small meals regularly; to avoid constipation; to avoid sudden movements; to avoid those who seem to have colds; and, should he catch cold himself, he should go to bed at once and treat it as serious. The patient should be encouraged to feel that the lesion in his heart need not prevent him from living a useful, happy life providing he forestalls repetition of the acute infections which increase the lesion, and providing he lives within the limit of his reserve strength.

CHAPTER VII

Diseases of the Respiratory Organs

Internal and External Respiration

All the living cells of the body need a constant supply of fresh food, which is sooner or later burned, with the production of ashes. For this, as for all combustion, **oxygen** must be present. Among the "ashes" produced in this process is carbon dioxide, a gas, and this with the other ashes must be removed from the cells as soon as formed; otherwise there is as much danger of the tiny cells' suffocating as there is of the whole man's dying if his oxygen supply is cut off and the carbon dioxide in his lungs cannot escape. This process of renewing the cells' air, or, more properly speaking, their oxygen, and of removing their "bad air," or their carbon dioxide, is called **internal respiration.** It is accomplished by the circulation of the blood. The cells (Fig. 52, *a*) are nowhere very far from a capillary, *b*, with walls so thin that gas and fluid can freely pass through them. In these capillaries is constantly passing a stream of red corpuscles whose hæmoglobin is saturated with oxygen. Between the cells and the capillary wall is a thin layer of lymph (practically the blood plasma). The tissue cells take from this lymph all the oxygen it has, and so the capillary is soon surrounded by a layer of lymph which has less oxygen than has the blood in the capillary. The oxygen therefore leaves the hæmoglobin and diffuses into the tissue lymph, where it is at the disposal of the cells. The cells produce carbon dioxide. This diffuses into the tissue lymph, and on into the blood plasma in the capillary, where formerly there was none. In this way the red arterial blood, rich in oxygen and free from carbon dioxide, changes in the capillaries to blue venous blood, rich in carbon dioxide and poor in oxygen. Venous blood must somewhere have the opportunity to get rid of this carbon dioxide and to take on a fresh supply of oxygen. This is accomplished in the **lungs,** the organs of **external respiration.**

The Lungs. In simple terms, a lung is an open sac filled with air. In its walls is a network of capillaries so constructed that gas but not fluid can pass easily through their walls. Some amphibians have such a lung. It consists of a hollow sac (Fig. 53), and a tube connecting the interior of the sac with the outer air. The sac has a thin wall on the outer side of which is a close network of capillaries. The wall is of such a nature that oxygen and carbon dioxide, but no fluid, can easily pass through. It is a law of gases to diffuse, that is, to distribute themselves uniformly throughout the whole of the space to which they have access. Set free a little bad-smelling gas in one room of a house, and soon it can be smelt in every room. If a large vessel is divided into two compartments by a partition of some porous membrane, and one compartment is filled with one gas, the other with another, both gases will diffuse through that membrane until each gas exerts the same pressure, or "tension," on both sides of the partition. The process of diffusion through a membrane is called "osmosis," and this is constantly going on in the lungs. There is much less oxygen in the blood flowing through the lungs than there is in the air; there is much carbon dioxide in the blood and very little in the air. These two gases therefore will rapidly diffuse in opposite directions until the blood has almost no carbon dioxide left and has gained much oxygen. There is a chemical attraction between oxygen and hæmoglobin, which makes them unite to form "oxyhæmoglobin." But this "affinity" is easily overpowered in the capillaries by a stronger affinity which compels the oxygen to pass by osmosis into the tissue lymph, where there is an oxygen vacuum. But some gases, for example the carbon monoxide of illuminating gas, have a stronger affinity for hæmoglobin than oxygen has. If a person breathes this, that hæmoglobin which has become "carbon monoxide-hæmoglobin" will not split in the

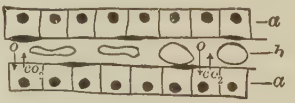


FIG. 52.—Internal respiration. *a*, tissue cells; *b*, capillary through which blood is flowing. (The lymph spaces are omitted for sake of clearness. See Fig. 29.) The interchange of oxygen and carbon dioxide is shown by arrows.

capillaries of the tissue or of the lungs and so is of no further use to the body; and hence if he breathes enough of this gas he might as well have lost just so much hæmoglobin, and he will die.

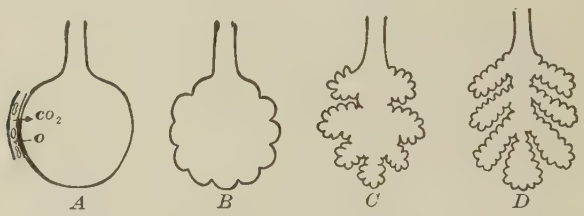


FIG. 53.—The simple lungs of amphibians. *A*, the simplest lung, showing a capillary in the wall and the interchange of oxygen and carbon dioxide. *B*, *C*, and *D*, are more complex lungs showing how increase in respiratory surface is gained without any increase in the bulk of the lung as a whole.

This simple lung (Fig. 53, *A*) is, theoretically, all that is necessary for a man, but, practically, it would not do, because we have so much blood to "oxygenate." A smooth sac would need to be huge to be big enough to enable the blood in our pulmonary capillaries to spread out in a thin layer one corpuscle deep. This difficulty could be met by crinkling and folding up this huge thin-walled sac into small volume, just as we might by folding, put hundreds of square yards of tissue paper in a small box. The low vertebrates solve this problem of furnishing sufficient respiratory surface by means of lungs shaped as in Fig. 53, B, C, D. A man's lung is really **a collection of myriads of small lungs,** each of which is separate and complete in itself. Fig. 54 represents one of our little lungs, which consists of a tiny bronchus, *a*, and many "air cells," or "alveoli," *b*. Understand this tiny lung, and you understand our big lungs. Were the respiratory surfaces of all these little lungs united in one big sheet, it would cover an area of 90

FIG. 54.—One of the tiny lungs of which our lungs are composed. *a*, terminal bronchus; *b*, air cells.

sq. meters. Were it made into one simple spherical lung it would be a balloon nearly 20 feet in diameter. The tiny bronchi are united like the twigs of a tree to form larger and larger bronchi (Fig. 55) till all the bronchi of each lung are joined into one primary bronchus, and the two primary bronchi then unite to form the trachea. For satisfactory respiration one thing besides mere surface is necessary. Since this bronchial tree has so many and so fine branches that it would take far too much time for the air in the alveoli to be renewed by diffusion from outside, it is necessary to expand them by pump-

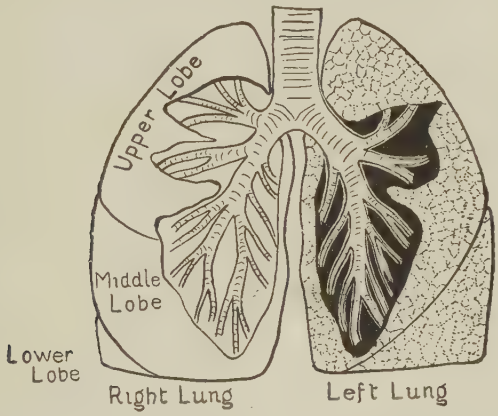


FIG. 55.—The bronchial tree. The two lungs are represented as opened, showing the branching of the bronchi. The mottling of the left lung suggests the tiny lungs (Fig. 54) which reach the surface (much exaggerated). The terminal bronchi and fine bronchi are not shown. These fine bronchi unite forming larger and larger tubes until there is but one for each lung, the primary bronchus. These two primary bronchi unite to form the trachea.

ing movements of the chest wall. When the alveoli expand new air rushes in which is forced out when they contract. The lungs are, therefore, put into an air-tight case with distensible walls, the chest. The only opening through which air can pass into the chest is through the trachea and on to the interior of the air cells. The chest resembles a pair of bellows. When we distend the chest a vacuum is produced. Air must rush in to fill up the vacuum, but it cannot reach the pleural cavity, which surrounds the lungs, so its only course is through the

trachea into the bronchi to the interior of the alveoli, which it distends, thereby inflating the lungs so that they still completely fill the chest. Then the chest collapses to its previous volume, and the elastic lungs collapse with it. In this way the whole lung is "ventilated"; that is, it receives by inspiration a supply of fresh air which is expelled by the next expiration. The lungs expand passively because they are elastic and follow the increased capacity of the cavity they must fill. Respiratory movements are made by the chest wall; their object is to make the cavity of the chest larger and smaller. The lungs collapse because they are elastic and always on the stretch. If a large cut be made through the chest wall the person could not breathe. The elastic lung would collapse to about one sixth of its normal volume, and then, as the chest cavity became larger and smaller, air would rush through the hole in the chest wall and in and out of the pleural cavity, not through the trachea into the interior of the lung.

Fig. 56. External respiration. *a*, the wall of a sac, consisting of one layer of cells, through which gases can pass, but not liquids; *b*, a capillary. The arrows show the interchange of gases.

Lungs consist of two structures, tubes and sacs. **The alveoli** (Fig. 54, *b*; Fig. 56), or lungs proper, are little air sacs about big enough to be seen with the naked eye. They have very thin walls made up of flat, epithelial cells. One side of these cells forms the inner surface of the alveoli; on their other side is a thick network of capillaries (Fig. 56, *b*). Through them gases can easily pass, but not liquids. The sacs' walls have great elasticity, and this quality allows them to be inflated by each inspiration. The tubes, or **bronchi,** (Fig. 54), have stiff walls, which do not expand and collapse. They are lined by a "ciliated mucous membrane" (Fig. 57). The epithelial cells of this membrane are not flat, as in the alveoli, but "cylindrical" and have their free ends covered with short hairs called **cilia,** which keep up a constant whipping motion,

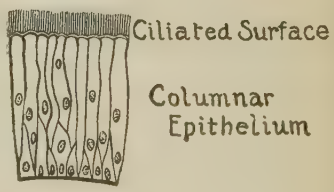


Fig. 57.—Ciliated epithelium (cross section).

always in the same direction, and thus sweep out any dust or excess of mucus on this membrane. Many notice that the first thing they do in the morning is to "clear their throat" and expectorate a little gray or black mucus. This did "come from their throat," but the sputum is the accumulated sweepings all the previous night of the whole bronchial tree, even to the base of the lungs. Some of that sputum is dust inhaled the day before; some is mucus from the air cells themselves,

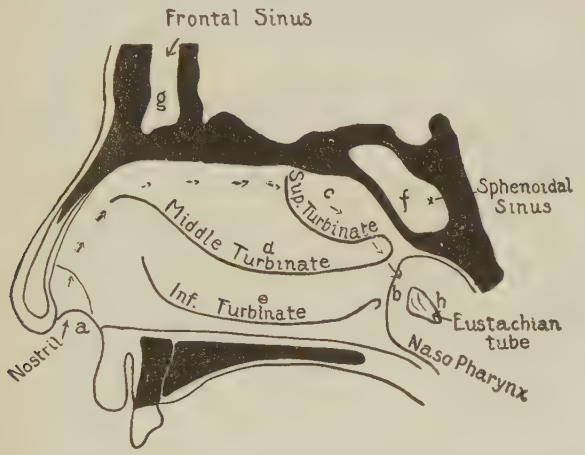


FIG. 58.—A vertical section through the nose. Black represents bone. *a*, anterior nostril; *b*, posterior nostril communicating with the nose and the nasopharynx or upper part of throat. *c*, superior, *d*, middle, and *e*, inferior turbinate bones. *f*, sphenoidal, and *g*, frontal sinuses. *h*, orifice of the Eustachian tube. The arrows show the direction of the current of air during respiration.

as well as from the bronchi; and in it are any germs which the cilia may have swept on. It is this sputum that we examine for tubercle bacilli in a very early case of consumption, before the "cough" begins. How hard it is to make our patient believe that there can be any significance in the particle of mucus which he "raises from his throat" every morning!

The Nose has two sides, which open externally through the anterior nostrils (Fig. 58, *a*) and posteriorly into the nasopharynx, *b*. These two passages are rather narrow at the front and back, but between they expand into large air chambers completely separated in the centre by the septum (Fig.

59, *a*.) The six turbinate bones (Figs. 58 and 59, *c*, *d*, and *e*) project into the nostrils, three from each side; the sinuses or accessory air chambers, open on the roof, at the rear, and on the sides (Figs. 58 and 59, *f*, *g*, *h*). **These sinuses** are the hollow interiors of the bones of the face. In the lower forehead, above the roof of the nose, are the **frontal sinuses** (Fig. 58, *g*); along the roof of the nostrils, the **ethmoid sinuses** (Fig. 59, *i*); opening at the rear, the **sphenoid sinuses** (Fig. 58, *f*); while the **antrums** (Fig. 59, *h*) open on the sides. All of these air chambers, the nose and the sinuses, open into the nasal passages and all are lined with the same ciliated epithelium.

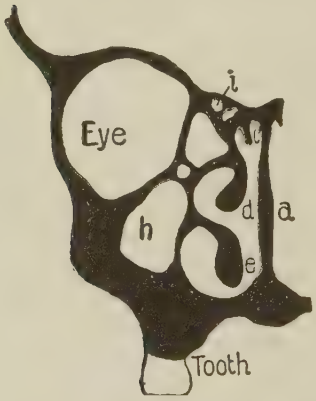


FIG. 59.—Cross section through the nostril (black represents bone). *a*, septum of nose; *c*, *d*, *e*, the superior, middle and inferior turbinate bones; *h*, the antrum; *i*, ethmoid sinuses.

The turbinate bones (Figs. 58 and 59, *c*, *d*, *e*,) which get their name from their shell-like appearance, are adapted by shape and position to increase the surface of the air chambers and to obstruct somewhat the air current. On the superior turbinated bone, *c*, and around it, is the membrane which is the seat of smell.

The air, on entering the anterior nostrils, **normally is considerably obstructed** by the turbinated bones and by other irregularities of the nasal wall, and so does not go straight through to the posterior nares, but passes upward to the roof of the nose, is reflected back and forth from side to side, and describes at least one circle before it reaches the nasopharynx. It therefore comes several times into contact with a great deal of mucous membrane. And this is intentional, for this **mucous membrane** is always rather wet with sticky mucus which **catches practically all the dust and germs** in the air that we inhale; its surface covered with mucus **moistens the air** to the desired degree and thus protects the lungs; it is very full of large blood vessels which **warm the**

air; its sensitive nerves **detect any unpleasant or dangerous odors**; the irritation of its surface **provokes a sneeze or cough** to expel any foreign body which comes into contact with it. The air, therefore, when it enters the nasopharynx, has been rendered suitable for the lungs; it is moist, warm, and almost free from dust and germs. But this is not the only important function of the nose. The air current is considerably obstructed by the tortuous passages of the nose, which **create just the right resistance** to the suction effect of inspiration and to the blowing effect of expiration. This is an essential element in the mechanism of respiration, but one not rightly appreciated until we see cases in which it is absent, as in mouth breathers. (See page 142.)

Diseases of the Upper Respiratory Organs

Bleeding from the nose, or "epistaxis," is common in young "full-blooded" persons. It may be due to injury or to the bursting in the nose of blood vessels which are unusually distended. It is very often an early symptom of typhoid fever. It is common in conditions of chronic passive congestion and often frequently gives very definite relief.

Acute coryza, or a "cold in the head," is a catarrhal inflammation of the mucous membrane of the nose. It is sometimes due to irritating fumes but usually to a bacterial infection of the nose. The germ is not yet known; in fact there are few diseases about which so little is known. The nose seems to be a point of least resistance, with conditions always present for the lighting up of an inflammation. We wet our feet, or a draught of air strikes our back. It is not often in the feet or the back that we suffer, but in the nose. In its mildest form and at the beginning and end of a severe case the mucous membrane becomes congested, turgid with blood, and secretes more of its ordinary mucous secretion than normally. This at first is a clear, thin, glassy, irritating fluid. Then the typical exudate appears; pus cells and perhaps also a few red corpuscles pour out from the capillaries, and the secretion becomes yellow. The swollen mucous membrane fills up the air passages, for normally there is little free

space in the nose, and a very slight swelling can entirely shut off nasal respiration. The orifices of the tear-ducts are in the nose and are closed by the swollen membrane, and so the "eyes run." The mucous membrane is now very sensitive, and hence we sneeze often to keep the nose clear from the exudate. An acute coryza is due to an infection. There are fever, and headaches, for a day or so. The exudation and the swelling of the membrane gradually subside during the succeeding three or four days, unless the trouble extends to the throat and lungs.

One should remember that an **acute coryza is often the first stage** of measles, of whooping cough, and of influenza, and should watch for the signs of these troubles in a child who has been exposed to them.

For treatment the patient is kept quiet until the acute stage is over. A calomel purge is given at once. Some local applications may relieve the trouble, especially local sprays.

Hay fever is a variety of acute coryza. It is more severe than a simple cold in the head; there is often bronchitis or true asthma with it; and the general symptoms—headaches, malaise, mental depression, etc., are more pronounced. The cause of this trouble is a nasal **mucous membrane more sensitive than normal** to certain irritating substances, as the pollen of plants (especially those of the rye family.)

Some persons have attacks in the spring ("June cold," "rose cold"), others in the fall ("autumnal fever"); and in regions without winter (for the first frost cuts short an attack) the hay fever may last all the year.

Among the hay-fever patients are some who have an idiosyncrasy to the **pollen of certain plants or odors from animals** (e.g. the dandruff of the horse); some whose noses are sensitive because of nasal infections; and some in whom the trouble is purely nervous.

There are persons whose **noses suddenly stop up,** sometimes without warning, sometimes as a result of some odor, a breath of cold air, etc. These patients may have some local nasal trouble, a spur or polyp in the nose, but sometimes this

is a signal of trouble further away, for part of the mucous membrane of the nose is a spongy tissue which, in response to various stimuli, including those from the pelvis, can suddenly fill with blood and become so thick that it closes the nostril.

The treatment of hay fever is, first, to cure any local trouble in the nose which may make this membrane too sensitive, and, second, to stay during the hay-fever season in some region where the victim is free from attacks. For most persons this is where they can breathe the dry mountain air. The hypersensitive nasal membrane can be destroyed, but the result may be worse than the hay fever. Many pollen vaccines have been used as a prophylactic for hay fever and with some, but not brilliant, success. The attacks of conjunctivitis, which are so troublesome a feature of hay fever, are best controlled by eye-drops and the asthma by hypodermic injections of adrenalin or epinephrine.

Chronic rhinitis. In acute coryza the nasal membrane may swell excessively, due to an excess of blood and a water-logging of the membrane with lymph. In its early stages all this can quickly disappear and the membrane return to its normal thickness. Later, however, the thickening is permanent. Many persons suffer from "chronic cold in the head," or chronic catarrh. They say that they "catch cold easily," but the truth is that they have the same cold in their head all the time, and that this cold occasionally flares up and then quiets down. In all these cases one suspects a pocket of pus either in the nasal passages or in the nasal sinuses and an abnormally large amount of connective tissue in the mucous membrane. This membrane and the bones become permanently thickened, and the result, when local, is a **spur** on the septum or a **hypertrophied turbinate.**

When a nasal infection creeps into one of the various sinuses or remains there after a general nasal infection, e.g. influenza, and sets up a **chronic sinusitis** the condition is more serious; for in the sinus the infecton may be active for months or years. This infection is most likely to occur if

the septum is so crooked (deflected) that the drainage of one nostril is difficult, since then a little swelling of the mucous membrane on that side will completely close the nostril and thus aid in the formation of local pockets of infection. If the trouble is in the frontal sinuses there is a continuous discharge from the nose; if it is in the sphenoidal sinus pus will trickle into the throat. These patients have morning headaches in the frontal or occipital regions and gastric disturbances from the pus swallowed. The constant irritation from the pus makes the mucous membrane grow thick, or hypertrophy, and also leads to polyp formation, to the growth of nasal spurs, etc. The lack of a visible discharge is not necessarily evidence that the sinuses are normal, and often the inspection of the nose gives no hint of the presence or amount of sinus trouble present. We therefore employ the assistance of transillumination and of X-ray pictures of the skull, which sometimes aid us considerably. The treatment is to open the sinuses in order that they may drain, and this may mean the local application of adrenalin with mild antiseptics, the cautery, or an operation. If the passages of the nose are narrowed on one side by a deflected septum, this should be straightened.

Another chronic infection of the nose leads to wasting, or **atrophy,** not only of the mucous membranes but also of the turbinate bones themselves. The result is that the nostrils become large empty caverns. The exudate, which in such a case sticks to the walls in large quantities has an exceedingly repugnant odor, which can be detected at a distance, and which makes conversation with the patient disagreeable. Fortunately for the patient, he smells nothing. This condition is called **ozæna.**

The tonsils. The nasopharynx (Figs. 58 and 60, h) is the upper part of the pharynx. Into it open the posterior nostrils, b, and the Eustachian tubes, i, from the middle ears. The most important and commonest trouble which occurs here is either the persistence or the hypertrophy of the pharyngeal tonsil, called also the **adenoid,** which should

be removed if it causes the least obstruction of the posterior nares or of the Eustachian tubes.

By tonsils we usually mean two almond-shaped bodies in the back of the mouth, at *K*, Fig. 60, but these, although the largest and most easily seen, are only part of a **circle of tonsils** which crosses the root of the tongue and the roof of the

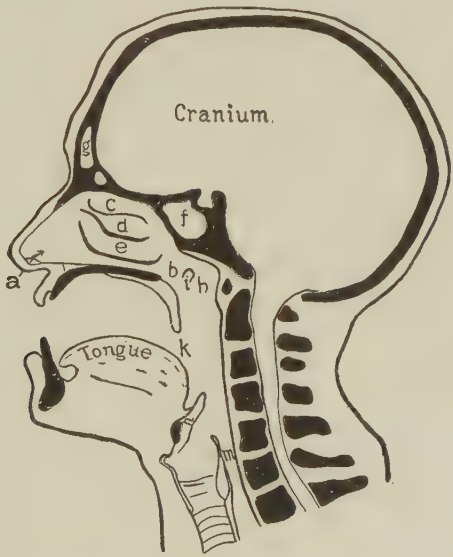


FIG. 60.—Vertical section through the skull. *a*, anterior nares; *b*, posterior nares; *c*, *d*, *e*, superior, middle and inferior turbinated bones; *f*, sphenoid sinus; *g*, frontal sinus; *h*, nasopharynx; *i*, Eustachian tube; *k*, position of tonsils; *m*, œsophagus; *n*, epiglottis over larynx.

nasopharynx, thus completely encircling the throat. The pharyngeal tonsil, or **adenoid,** at the roof of the nasopharynx is always present in children but normally soon disappears. If it becomes unusually large it may completely fill up the nasopharynx and be felt or even seen hanging down behind the soft palate (Fig. 61). This tonsil is just as dangerous a portal of entry for infection as are the throat tonsils. That phase of the subject we discuss under the heading Acute Tonsillitis (page 506). We are now speaking of the trouble

which **the adenoid,** filling the posterior nares, can cause by its presence. It **is responsible for a surprisingly long list of disturbances.** It obstructs the nasal respiration at an age when the bones of the face and chest are growing, are soft, and hence are easily influenced. The base of the nose becomes broad and prominent, the nostrils narrow. The eyes are far apart. The expression is rather stupid. The hard

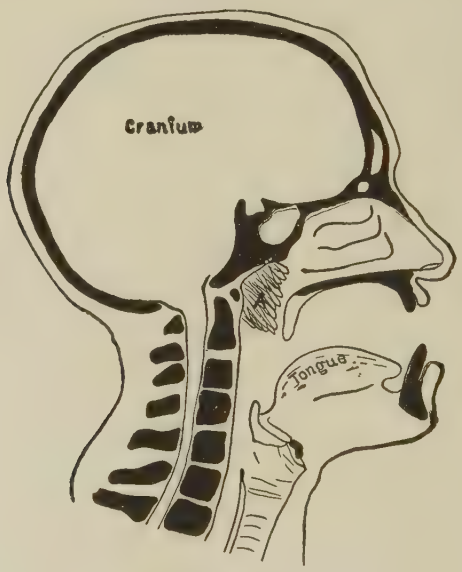


FIG. 61.—Vertical section through the skull showing a mass of adenoids, *A*, in the nasopharynx.

palate of the mouth becomes high-arched; the upper jaw becomes narrow; the upper front teeth project, "rodent fashion"; the voice is nasal. The sternum becomes prominent (pigeon breast); the lower margin of the chest is hollowed (Harrison's groove). It is strange that parents do not realize that the difference between some very pretty and some homely faces, between well-shaped and deformed chests, is due to adenoids, and that the child's beauty could have been preserved. The adenoid also blocks the orifices of the Eustachian tubes. The result is often chronic otitis media with

slowly developing deafness, and sometimes acute suppuration of the middle ear—"running ears"—with the blood poisoning, the abscesses, and the bone and joint inflammation which may follow.

The pharynx, the larynx, and the lungs suffer from an adenoid, because the air is not so well warmed and saturated with moisture, not so thoroughly freed from dust and germs, by the mouth as by the nose, and chronic laryngitis and chronic pharyngitis are the logical result. The child with an adenoid does not sleep well but has bad dreams, wakes suddenly with a "night cry," and is found sitting upright in bed. A more important result is asthma, and this serious trouble may continue throughout life, even after the adenoid has been removed or has disappeared. The result of asthma is emphysema of the lungs, a serious condition. Lastly, for some reason which we don't know, children with adenoids do not develop mentally so well as other children. They not only look stupid, because they are mouth breathers, but they sometimes really are stupid. These adenoids can be easily removed by a simple and satisfactory operation.

The Larynx and Trachea. The nose and nasopharynx are lined with the same kind of ciliated epithelium as the larynx and whole bronchial tree, but the pharynx, or throat, the place where the respiratory and alimentary passages cross each other (Fig. 60, m, n, and Fig. 62), is lined with "flat," "squamous" epithelium. This is fortunate, since the inflammation of epithelial surfaces has a tendency to spread. A bronchitis of the finest tubes will quite certainly spread up to the nose; a coryza will tend to spread down to the bronchi. But this strip of epithelium of a different character acts as a barrier, and an acute coryza does not always spread to the throat, an acute bronchitis seldom spreads to the nose. **An acute inflammation of ciliated epithelial membrane** is similar to that described as coryza, whatever part of the respiratory tract is affected. We have, first, hypersecretion of a clear mucus, then a mixture of pus and red cells, then a profuse discharge of watery pus, then

gradual diminution of the leucocytes till the abundant but still diminishing exudate is pure mucus, then the disappearance of this also. The membrane is first moist, then red; the epithelial cells are often cast off in large sheets, leaving the bleeding, suppurating submucosa bare; then this gradually becomes re-covered with epithelial cells. When this process affects the larynx, we have **acute laryngitis**; when the trachea, **acute tracheitis**; when the bronchial tree, **acute bronchitis.** But there are differences in symptoms; these

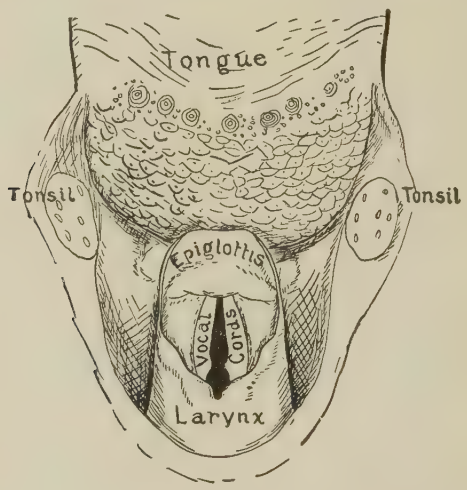


Fig. 62.—The throat (viewed from above and a little behind.)

vary according to the location of the process. **In acute laryngitis** speech is difficult, and the cough is especially severe. The cough is relieved by resting the voice, by breathing air moistened with steam, by an ice bag on the throat, and by local soothing sprays. Chronic laryngitis is due to repeated attacks of the acute trouble, which is caused by chronic sinus trouble above, by the frequent inhalation of irritating gases, as tobacco, and by habitual overuse of the voice, in the case of the street huckster.

Œdema of the larynx is a very serious condition, for the

patient may suffocate. The larynx is a stiff box of cartilage and will not stretch. Its orifice, the space between the vocal cords, which is called the **glottis** (whence the synonymous term œdema of the glottis), and through which the air must pass, is really rather narrow, and a little swelling of the mucous membrane may close the orifice tightly. This occurs rarely in acute laryngitis, less rarely in severe inflammation of the throat, such as is present in diphtheria, erysipelas, scarlet fever, and often when there is an ulcer there, as in tuberculosis, typhoid fever, etc. It also is often the cause of death in Bright's disease. In some patients with "giant œdema," local swellings due to œdema and of nervous origin, this may be the point which swells. **The patient with œdema of the glottis** suddenly becomes more and more dyspnœic; he struggles to draw each breath; the voice is at first husky, then lost. Unless an ice bag is put over the throat or a spray of adrenalin applied, or, these failing, the throat is opened below the larynx, the man may die in an hour or so. If the operation of tracheotomy had been known in George Washington's time, his life might have been prolonged.

An attack somewhat similar but of very different nature occurs in neurotic children under three years of age, especially in babies with rickets. It is due to spasms of the muscles of the throat. The child struggles for breath; the face becomes blue. Then suddenly the spasm is relieved, and the child takes a deep breath with a crowing sound. This condition is called **laryngismus stridulus,** or, in popular terms, "child crowing" or "passion fits." It is a nervous trouble and should cause no alarm. The general condition of the child should be built up.

Spasmodic croup is thought to be a similar condition. The child awakes at night with a sudden difficulty in breathing, a croupy cough, husky voice, and congested face. In a short time the breathing is suddenly relieved. This may occur for several nights. During the day there will be a mild

bronchitis. Various remedies will relieve the spasm—a warm bath, an emetic, a whiff of chloroform, etc.

The larynx is often attacked by tuberculosis, and this condition is very serious. Patients so affected are hoarse, have the worst cough of all, and suffer terribly when they try to

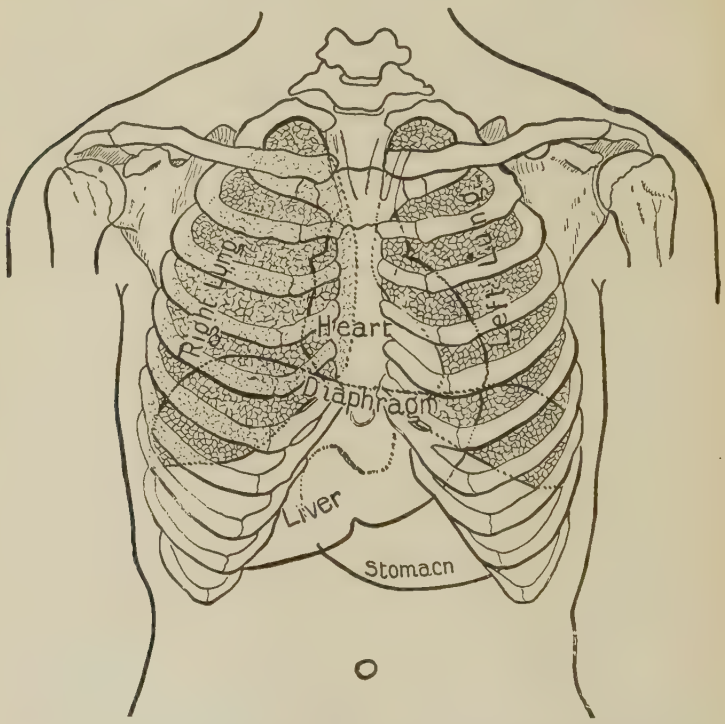


FIG. 63.—A diagram of the normal thoracic and upper abdominal organs.

swallow. This suffering, however, has been much less recently, since the introduction of more successful treatment of this condition, with complete rest of the infected parts. Cancers of the larynx are common and require the removal of the entire thyroid cartilage.

DISEASES OF THE LUNGS

Acute Bronchitis, or a "cold in the chest," is an acute in-

flammation of the mucous membrane of the bronchial tree. It may extend down from the nose or from the larynx, or may begin as a cold "on the chest." It may be an early symptom of more severe diseases, such as measles, whooping cough, typhoid fever, or influenza. The "cold on the chest" of adults is usually a laryngo-tracheitis only, the infection scarcely reaching the large bronchi. In children, on the contrary, such inflammation often extends into the air cells and sets up a broncho-pneumonia. The symptoms of acute bronchitis are partly general, that is, fever, headache, malaise, languor, various general pains; and partly local, such as a cough, painful and dry at first, then easier and loose, with abundant sputum. The course of a simple case is about seven days. The treatment, apart from the various medicines which may be prescribed, is rest in bed till the fever is all gone, warm baths, and a mustard plaster or water jacket on the chest. An ordinary kettle boiling in the room will keep the air moist and may make the patient more comfortable. A tendency to recurring attacks of acute bronchitis always means that the patient has also some focal lesion from which this infection spreads. Frequently, infected nasal sinuses are such foci and should be treated accordingly. More often these sinuses, especially the sphenoidal and posterior ethmoidal, infect first the lymph nodes along the trachea and especially those at its bifurcation and on the primary bronchi, therefore in the hila of the lungs, causing the mediastinal adenitis so common since the influenza epidemic. It is to the infection of these glands that many recurring attacks of bronchitis and bronchopneumonia are due.

Chronic bronchitis is one of the most common and troublesome of maladies, especially for the aged. It is the result of recurring acute infections (especially those which spread from the nose or from the mediastinal lymph nodes). After years, as a result, the mucous membrane of the bronchi finally becomes thicker or thinner than normal is often very sensitive. Patients so affected have a "chronic cough"—some a "winter cough," some a "morning cough." Some

raise almost no sputum; some, vast amounts of a watery sputum; some, a very fetid sputum ("putrid bronchitis"); some expectorate only in the morning and "clear their tubes for the day." But chronic bronchitis may be either a complication or a symptom of a more serious trouble, such as chronic lung disease (e.g. emphysema) or Bright's disease or gout, etc. It is very common in mitral heart disease because the years' long, chronic, passive congestion of the lungs greatly predisposes them to infection.

Patients with chronic bronchitis should first have any chronic infection of the upper respiratory tract treated, then should choose that climate in which they have least trouble. They should so dress as to avoid catching fresh colds. Among drugs, potassium iodide is especially helpful in relieving the dry cough.

Fibrinous bronchitis is a remarkable condition in which the bronchial walls excrete fibrin. The characteristic symptom is the expectoration of treelike casts of the bronchi.

Asthma, to the popular mind, means recurring attacks of shortness of breath. But these may be due to diseases of the heart ("cardiac asthma"), of the blood vessels, or to Bright's disease ("renal asthma"). In true asthma the trouble is in the finest bronchi. These, either because of swelling of the mucous membrane (these little tubes are so fine that a slight swelling will almost close them) or because of spasm of the muscle fibre in their walls, may become so narrow that air can scarcely pass through them to the air cells, or, to speak more properly, can scarcely be squeezed out of the air cells. In cases of true asthma, although each inspiration is very difficult, each expiration is still more so. The patient **tries to squeeze the air out** of his lungs by forcibly contracting the muscles of expiration and makes **a loud wheezing sound** when doing this. It is certain that asthma is partly a nervous disorder, and for this reason one form of it is called "hay fever of the lungs." The most typical cases are a part of hay fever or are equivalent to it. They come at the same season and respond to the same treatment. But with asthma there is

often bronchitis, that is, an inflammation, so that we are sure it is not entirely a neurosis.

In many cases asthma has a "reflex" origin; that is, the real disease is somewhere else but through the nervous system produces its symptoms here. For instance, a person often has bronchial spasms wrongly called asthma as a result of an adenoid, or he has asthma because he has a polyp in his nose. In such a case remove the adenoids or the polyp, and the asthma will disappear. Other asthmatic attacks are "touched off"—for they resemble a convulsion in many ways—by some trouble in the stomach or in the pelvis. Certain odors, a cold breath, dust, perhaps certain foods, may bring on an attack; but all theories which postulate some proteid poisoning or sensitization, the so-called anaphylactic reactions, are so theoretical that they tend to divert our attention from the primary neurosis or the nasal disease which these patients certainly do have.

Asthmatic attacks come on suddenly, usually at night. The patient feels chilly, has a tight feeling in the chest, and has difficulty in breathing. He sits up in a chair, looks anxious, and begins to perspire freely. He takes slow, deep breaths, struggles to draw the air into the lungs, and works still harder to blow it out. The respirations are loud and wheezing. This lasts a few minutes or hours, and then comes relief; but there will still for a few hours or days be some wheezing, and a bronchitis will develop, with cough, and an expectoration in which can be found the prettiest spirals of twisted mucus that one ever sees. These attacks may come on night after night or only on very rare occasions. The results of them in time are serious, for the lungs become emphysematous because of the chronic bronchitis which has been present, and even the shape of the chest changes.

The cause of asthmatic attacks must be found and removed. This means that the nose, the throat, the pelvis, and the genital organs must be carefully examined and any harmful conditions corrected. The patient soon learns not to eat heartily or late at night and to avoid cold draughts and

any odors, etc., which precipitate the attacks. He usually keeps himself armed with "perles" of amyl nitrite (the same as used in angina pectoris) or a small bottle of chloroform or paper steeped in potassium chlorate, belladonna or henbane, the fumes of which, when burned, will relieve the dyspnœa. Between attacks potassium iodide is very helpful.

Emphysema of the lungs is a condition easily understood if one remembers that our lungs are a myriad of tiny lungs, each about 1/100 inch in diameter, with elastic walls, which are inflated to larger size by each inspiration, and which because of their elasticity collapse partly during each expiration. Suppose now that the walls are naturally not very elastic because they are abnormally weak or are repeatedly overdistended. They will, then, become less and less elastic and will during expiration remain inflated; their weak walls will become weaker until some unusual respiratory movement will burst them, and two or more air cells will then become one large one. Thus the process continues. **The lung becomes less elastic and more and more inflated**; more and more of the **air cells break down**; the air cells become larger until some are 2 or 3 mm. in diameter; and in places the lung contains cavities even as large as a hen's egg. Fig. 64 shows, below, air cells of normal lung cut across, and, above, those distended as in emphysema.

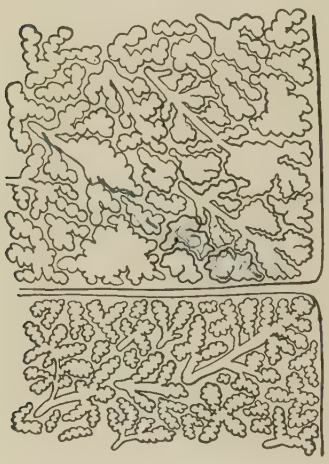


Fig. 64.—Emphysema of the lung. Above is a portion of a very emphysematous lobe of the lung. Below is represented for comparison a portion of a normal lobe.

In emphysema each breath requires an unusual amount of muscular force, for the patient must force the air out of the lungs. Inspiration is short and quick, expiration long and wheezing. He will, therefore, be "short of breath" between the asthmatic attacks. The chest will become thick and bar-

rel shaped. Another result is that there is much less respiratory surface because so many walls of alveoli have disappeared, and, of course, their capillary network also. Patients so affected always look blue, or "cyanotic." It requires an unusual amount of work for the heart to pump the blood through such lungs, and hence the right side of the heart

FIG. 65.—Emphysema of chest in young girl.

hypertrophies. And, finally, there is, most of the time, considerable bronchitis.

Emphysema might be expected to develop in the lungs of men who do a great deal of hard blowing, as horn players, glass blowers, and those whose daily work is hard muscular labor. But in these men the emphysema is seldom of as severe a grade as that occurring in the children with adenitis

or in adults with asthma. Indeed, the worst cases may be best explained by the inheritance of poor lung tissue.

The best treatment for emphysema, as for asthma, is to remove the cause—the adenoids, etc. Patients with emphysema will of necessity lead a quiet life. The chronic bron-

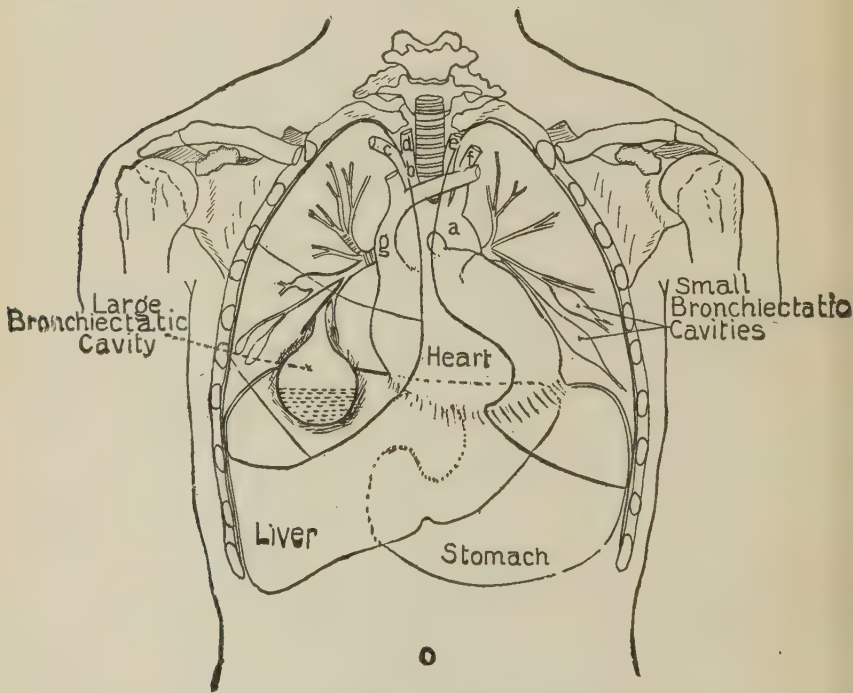


Fig. 66.—Lungs the seat of bronchiectasis. *a*, aorta; *b*, innominate artery; *c*, right subclavian artery; *d*, right common carotid artery; *e*, left common carotid artery; *f*, left subclavian artery; *g*, superior vena cava. In this figure the pericardium is represented as not opened.

chitis to which they are especially susceptible needs treatment.

Bronchiectasis (Fig. 66) is a condition usually the result of chronic bronchitis. The bronchi are tubes with walls made rigid and strong by rings of cartilage. When because of disease these walls become weak, the lumen of the tube becomes dilated, and the development of a sac-like cavity may

take place. These cavities differ from those of tuberculosis, for their walls are those of the bronchi. These cavities are distended bronchi, while in tuberculosis there is death and decay of lung tissue, the expectoration of which leaves a hole behind. Sometimes in bronchiectasis one or a few bronchi become much dilated, a condition known as "saccular bronchiectasis." If a great many bronchi become just a little dilated the condition is called "diffuse" or "cylindrical," bronchiectasis. Many cases date back to an attack of grippe. But not only must the bronchial wall become weak, but there must be a force to inflate the bronchial cavity. Repeated coughing can do this; the accumulation of exudate in the growing cavity can by its weight do this; and the bands of scar tissue which in pulmonary tuberculosis radiate from the bronchi can by contracting separate these walls. The symptoms are those of chronic bronchitis unless one or more cavities are very large.

These large cavities may hold a cupful or even more than a pint. The sputum collects in them, and on some change of posture—usually on rising in the morning—a little sputum flows out into a normal bronchus. Then the patient coughs until he has emptied this sac. **The expectoration of large amounts of sputum, in paroxysms,** usually means saccular bronchiectasis. Patients so affected often have hæmorrhages from these cavities, sometimes very severe, and the odor of the sputum is often very foul. If there are many small cavities there may be no symptoms to distinguish this condition from chronic or putrid bronchitis.

The treatment is that of bronchitis. The odor of the sputum can be much diminished by creasote inhalations. Out of a sheet is made a tent, beneath which is a chair on which the patient sits. Under the chair is a small lamp which warms a basin of water containing one teaspoonful of creasote. The fumes, inhaled for twenty minutes or an hour every day, relieve the cough and make the odor of the sputum much less objectionable. Another and more successful treatment is the injection of medicine directly into the trachea

through a syringe, the point of which reaches the larynx. The large cavities are helped to drain by postural exercise and may if suitably located be emptied through a bronchoscope. When they are very large the best therapy may be to cut out parts of the ribs behind and in front of the cavity and then to collapse the affected lung by an amount equal to the size of the cavity.

5 **Chronic passive congestion of the lungs** is mentioned on page 120 in connection with heart lesions, especially mitral stenosis, but as a pulmonary condition alone it deserves attention, since it gives rise to symptoms which should be recognized, and whose causes should be treated. Patients with this trouble tend to be short of breath. They have a chronic cough with a large amount of **sputum, often streaked with brown pigment** (altered blood), are susceptible to attacks of acute bronchitis on very slight provocation, and are subject to hæmorrhages from the lungs, some of which are profuse and yet are followed by great relief. It may be that the correct diagnosis of this pulmonary condition will lead to the discovery of the heart lesion which causes it, but which until that time has not been suspected. The treatment, of course, is that of the heart disease.

6 There is another pulmonary congestion, which is even more serious than general chronic passive congestion, and which involves the lower lobes of very weak patients with chronic illnesses, especially long fevers, who are allowed to lie for long periods on their back. It is called **hypostatic congestion** because gravity seems to be the determining factor in its production. Gravity is important but could not produce it alone, since a more normal person can even for months remain in a horizontal position without ill effects. In its severe grades and aided by infection it becomes a true pneumonia, called **hypostatic pneumonia.** The treatment of hypostatic congestion is chiefly the treatment of the heart condition. Beyond that it is a nursing problem. The patient should be turned frequently from side to side, encouraged to take deep breaths, and, in some cases, bled.

Pulmonary œdema, that is, the passage of blood plasma directly into the air cells, is an example of the most severe congestion of the lungs. In the air passages this fluid, mixed with the air and churned by the respiratory movements, produces the so-called "death rattle," a multitude of coarse, crackling sounds from the chest. In a later stage of the œdema this frothy mixture of air and blood plasma is expectorated in large amounts as sputum, and still later it may flow in streams from the mouth and both nostrils. Œdema of the lungs occurs in acute form in pneumonia; it sometimes follows the too rapid removal of a pleural effusion; but most often it is due to heart disease. Some mitral cases are subject to frequent transitory attacks of pulmonary œdema, which begin with a sense of oppression and pain in the chest, and the next symptoms of which are dyspnœa and orthopnœa, followed by cough with an **abundant blood-streaked foamy sputum.** In most cases, however, it is an incident of the last hours of life, when the left side of the heart fails, such failure occurring especially in patients who for years have had heart disease, although a larger number had not been considered heart cases but cases of chronic nephritis, or arteriosclerosis, or of essential arterial hypertension, conditions in which the heart suffers as a result of a more general disease.

The treatment of the transitory cases of pulmonary œdema is rest, light diet, digitalis, and, in milder cases, atropin. We stimulate the heart as much as we dare, to help it carry its load, and often find that venesection is very useful in easing it. For the terminal cases there is no treatment.

Hæmorrhage is another result of **congestion of the lung.** We have mentioned this in connection with chronic passive pulmonary congestion. We shall mention it later in connection with tuberculosis, pneumonia, and other diseases with a general tendency to hæmorrhage, as purpura, etc. In another group of cases it is due to aneurism of the arch of the aorta, the pressure of which against a bronchus may cause ulceration of the bronchial mucosa, but which in more cases

slowly perforates into a bronchus, the blood for some time leaking through the lung. Finally, there are some healthy young persons who are suddenly terrified by coughing up blood. While the majority of these cases are tuberculous, some of whom may get well before the diagnosis is made, there are a few others the cause of whose hæmoptysis is never determined. The treatment is to keep these persons, in whatever group they belong, perfectly quiet, to encourage them, and to give opium in large doses if necessary.

9 Pulmonary infarction is still another cause of hæmorrhage from the lungs, and one seen chiefly in cases of heart disease. Infarction in general is described on page 89. That of the lung is due to emboli which come from the cavities of the right heart or from some of the veins of the body, especially those in the pelvis, or from the seat of recent operation. This is one of the least understood, most sudden, and most dreaded of post-operative complications. Once a branch of a pulmonary artery is plugged, there immediately develops an infarcted area which quickly becomes a red (hæmorrhagic) infarction, made soggy because of the blood which escapes from the capillaries of the affected area. The large emboli, some of which were probably thrombi from the first, cause the very sudden deaths so much dreaded by surgeons. The first symptom of a smaller embolus is a sudden pain in the chest, due to a local pleurisy over the infarcted area, and the coughing of a sputum stained by streaks of bright red blood. It should be emphasized, however, that fairly large branches of the pulmonary artery can be plugged without leading to any infarction. This can develop only if the lung previously has been the seat of chronic passive congestion. Of course there is no direct treatment. One merely keeps the patient quiet and comfortable.

10 Abscess of the lung is usually the result of an infected infarct. This, we infer from the study of the X-ray plates after operations, is much more common than we had supposed, and the lung seems to have the ability to heal without much of a scar rather large cavities, providing the germs

are not too virulent. The centre of an area of pneumonia also may become an abscess, while of course an abscess in the liver or pus in the pleural cavity or suppurating mediastinal lymph nodes perforating through the lung may also cause an abscess of the lung. An abscess in the liver, the pleura, or a mediastinal lymph node may, however, ulcerate through into a bronchus and be expectorated without causing a lung abscess. What happens is that the lung between the extrapulmonary abscess and a bronchus first becomes well consolidated by the infection before the formation of the fistula through into the bronchus.

An abscess causes a cavity in the lung, small or large, the walls of which are the necrotic, broken down, lung tissue itself, and the contents of which have escaped through a bronchus. The best treatment of an abscess of the lung is to teach the patient to lie so that the abscess will drain easily into the bronchus. The proper position may be determined by X-ray pictures. Sometimes we are able to suck the contents out of the abscess cavity through a bronchoscope. If the cavity is so large and its walls so firm that spontaneous healing seems impossible, it may be necessary to collapse that side of the chest sufficiently to obliterate the abscess cavity. This we do by removing an inch, both in front and behind, of each rib over the abscess cavity.

Areas of infarction, if infected by the germs of gangrene, can break down, producing cavities the contents of which have a horridly foul odor.

Fibrosis of the lung is a chronic condition which slowly shrinks this organ and makes it immovable on respiration, drawing the ribs so closely together that the interspaces are obliterated and pulling the shoulder down and the spine over towards that side. A local fibrosis, as we shall see later, develops in every case of pulmonary tuberculosis in which there is sufficient resistance to the disease to allow the formation of considerable scar tissue. A more general diffuse fibrosis of whole lobes or of one whole lung may follow a pneumonia which did not resolve properly. But more important

causes are chronic infections of the pleura or of the mediastinum. These lead to the formation of dense bands of scar tissue which penetrate the lungs, and which later, as does all fibrous tissue, contract. In these cases especially are bronchiectatic cavities apt to develop, because the walls of the bronchi are pulled apart by the contracting scar tissue around them.

A slight grade of **fibrosis** develops in the lungs of those who inhale a great deal of dust, as coal miners, those in the grinding trades, etc. This dust must be deposited in the lymphatic spaces around the bronchi, and this leads to the formation of a good deal of scar tissue around these deposits, so that, when the scar tissue is widespread, the lung may become uniformly thickened, although not to an extreme degree. The condition is called pneumonoconiosis or, literally, **lung-dust disease.** In the case of the miners it is called anthracosis.

Pleurisy is inflammation of this very smooth, slippery membrane called the pleura, which covers the interior of the chest cavity, the upper surface of the diaphragm, and the outer surfaces of the lungs. The pleura is always **moistened by a lymph,** so that in the respiratory movements these surfaces can rub freely and painlessly against each other. That on the lung is called the visceral pleura; the rest, the parietal pleura. If a small area of a pleura surface becomes infected, the respiratory movements at once become very painful, and the patient holds that side as firmly as he can and takes the shallowest breaths possible, in order to diminish to the minimum extent the area over which these two surfaces will rub together. Since the inflamed areas will soon be covered by a thin sheet of fibrin, this disease is called **fibrinous pleurisy.** This trouble often begins with a chill and fever, a sharp and cutting pain often called **a stitch in the side**, and a cough which is short, very painful, and without sputum. The trouble may last only a few hours or days. While such a pleurisy can easily follow any deep wound which penetrates the chest wall, by far the majority of these cases are tubercu-

lous and often the first evidence of this disease the patient has. The treatment is to immobilize the affected side as completely as possible, which can be done by **strapping it with broad strips of adhesive,** or, if the pain is in the lower part of the chest, we can tie a tight bandage around the thorax, immobilizing it, and allowing the patient to breathe with his upper chest. Cupping, blistering, mustard plasters, heat externally applied, the Paquelin cautery, in fact any counterirritant, are helpful in the more chronic cases. In general, morphia should be used frequently and the patient kept in bed as long as there is any fever, and in the future treated with a view to preventing any further development of tuberculosis.

Pleurisy with effusion sometimes, not always, begins with a fibrinous pleurisy, and, often when it does, the pain is so slight that the patient scarcely feels it or quickly forgets it. What usually happens is that he gradually becomes paler, short of breath, easily fatigued; he loses weight and strength and sometimes, not always, has a little dry cough. Many a person works unconscious of disease until too tired to continue, when the doctor examining him is surprised to find that one of his pleural cavities is almost full of fluid. On inspection this side looks definitely distended, does not move on respiration, is flat on percussion; the heart is visibly moved to the more normal side, and the normal lung is seen to make visible efforts to breathe for both sides. The condition is clearly demonstrated on X-ray plates of the chest. Some of the fluid should at once be obtained through a hypodermic needle for examination. In this disease it will be found to be clear and straw-colored and to clot on standing in the jar. It is practically blood plasma. Nearly all of these cases of pleurisy with effusion are **due to a tuberculosis of the pleura** and have the same significance as a fibrinous pleurisy. The treatment is to remove enough of the fluid through a large needle to keep the patient comfortable. Formerly, the chest was tapped repeatedly in order to keep the pleural cavity as dry as possible, but this is no longer thought desirable, since the fluid

probably has some protective value and so will reappear if removed too early and will disappear spontaneously at the right time. Whether to tap early and much, however, is still an undecided question. The patient should be kept in bed as long as there is any fever and receive nourishing food and cod-liver oil. The nasal sinuses should be treated, infected tonsils removed and any other infection of the body eliminated which could stir up a latent tuberculous lesion. The patient should in the future guard himself against further progress of tuberculosis.

Not all fluids in the pleural cavity are due to tuberculosis. **In heart cases** with bad compensation both pleural cavities, especially the right (since the dilated heart obstructs the circulation in the hilum of the right lung far more than it does that of the left), may contain fluid. **In cases of nephritis** even when general dropsy is absent **the pleural cavities may contain a good deal of fluid.** In these and in the heart cases, however, the fluid is thinner than in pleurisy with effusion, is more watery, and will not clot on standing, and therefore is called a transudate rather than an exudate, although the difference is rather theoretical. The treatment of these cases is that of heart disease or renal disease.

If the pleura contains pus instead of serum, the condition is called **empyema of the chest.** Then the problem is quite different. This condition like pleurisy with effusion is often latent. A patient so affected looks much sicker, has a higher fever, and is more anæmic than are cases of pleurisy with effusion. It is very seldom that the pus is present in as large amounts as is the clear fluid. In addition to these cases are others due to tuberculosis, in which case the fluid at first may have been clear and later made purulent by a secondary infection; and others which follow a pneumonia, or an abscess of the lung, or of the liver.

Whenever fluid is suspected in the chest, or is demonstrated on X-ray plates, **a needle should be plunged** through an intercostal space in order to determine the character of the fluid, and if it is pus its removal should be considered

much more seriously than if it is clear. Much will depend on the germ present. If operation is decided on about two inches of a rib is resected (so that the wound may not close too quickly) and an incision made into the pleural cavity, allowing free drainage of the pus. During the past few years the so-called "closed treatment" has been tried with the use of irrigations of Dakin's fluid, and in suitable cases with success.

The treatment, following the resection of a rib, is of course chiefly nursing, nourishing food which is easily digested, the ultraviolet light, etc. During the convalescence breathing exercises and gymnastics may be of value in order to keep the lung of the affected side distended and to prevent its too great collapse.

Chronic pleurisy is the result of an acute pleurisy or of an empyema inadequately treated. The two pleural surfaces becomes firmly united, thus **obliterating the pleural cavity,** and the pleural membranes grow thicker and thicker, thus compressing the lung, preventing its expansion, invading its tissue, and finally almost destroying it. The diseased side of the chest becomes immobile and shrunken, the shoulder is pulled down, the spine bent over to that side, and the patient is generally miserable. It is not necessary that this should happen. So large amounts of scar tissue usually mean that some pus, which might have been drained out, was allowed to remain inside the pleural cavity; also perhaps that the proper after-treatment was not administered.

Pneumothorax means the presence of air within the pleural cavity. The result is that the lung, an elastic organ which is normally considerably distended, collapses. The air may get in through an external wound, although the external air rarely enters, since nature is skilful in preventing this complication. More often the pulmonary pleura also was wounded, and so it is air from the trachea which enters the pleural cavity. Sometimes a fractured rib may wound the visceral pleura. The majority of the cases of pneumothorax, however,

are due to the **rupture of a small tuberculous area** just beneath the pleura or to the rupture of an emphysematous bleb. Without any warning patients with these lesions suddenly find themselves short of breath. The ruptured side will appear full and quiet on respiration, its intercostal spaces will bulge, the heart will be pushed over to the other side, and the liver will be pushed down. Sometimes we must allow a part of the air to escape, so high is its pressure, but in most of the cases we leave it alone. As the reader will learn later, in the treatment of **pulmonary tuberculosis,** we often **create** a pneumothorax artificially by pumping air into the pleural cavity of the affected lung in order to keep this lung quiet, and so we think of **pneumothorax as in part a beneficial process.** As a rule, however, fluid also, due to a secondary infection of the pleural cavity, is present in the pleural cavity, and the condition is one of **hydropneumothorax or pyopneumothorax.** The splashing of this fluid in a pleural cavity containing air may make a beautiful musical ring which can be heard for some distance, and which is called the **Hippocratic succussion splash** because Hippocrates first described it. Of course we should in these cases remove the pus just as in cases of empyema. The air alone, we feel, may help the patient. Otherwise, the treatment is that of cases of pulmonary tuberculosis.

Tumors of the lung are not rare, since the bloodstream will bring to the lungs cancer nodules from any primary tumors elsewhere in the body. These grow in and between the alveoli, which they push apart in their growth, and their presence **may cause no symptoms,** unless they are very large; in this case there is merely some shortness of breath. If, however, a nodule grows just under the pleural membrane, the affected side of the chest becomes filled with fluid which quickly collects again when removed. Sometimes cancers start in the lung itself, that is, in the mucous membrane of the bronchi, and these cause many symptoms. At first they may plug the bronchus, and that causes a cough that is very

difficult to control; then, like other cancers, they soon break down and bring about the formation of a large cavity in the lung, which closely resembles an abscess of the lung or an empyema. In these cases all we can do of course is to keep the patient comfortable.

CHAPTER VIII

Food Facts

Food and Foodstuffs

By **food** we mean substances necessary to the normal composition and functioning of the body. Withdraw water, salts, and the other food substances, and the body will die.

The word "foods" suggests to us the various meats, grains, vegetables, fruits, etc., which we eat. These, however, are not simple substances, but are mixtures of **foodstuffs,** and these in turn are classified as **proteins, carbohydrates** (sugars, both the simple monosaccharides and the complex polysaccharides, and starches), **fats, salts, water, and vitamines.** A piece of meat, for illustration, contains all these food stuffs. The multitude of the proteins vary according to their origin, taste, and appearance. Chemically they are somewhat similar; and biologically they were originally the living protoplasm of the animal, or plant, from which they came. Though the various vegetables and fruits contain many proteids, and many different carbohydrates, and the oils and fats of no two animals, or vegetables, are exactly the same, chemically all these foodstuffs may be classified under the five headings given above.

Another division of foods—a physiological division—may be made between those **foods which the body uses as fuel,** and **those with which it repairs** its used-up protoplasm. The foods of the first group, and what is here said is also true of sugar and fat, are used as coal is used in a furnace, that is, to produce heat. The foods of this group may be burned at once, or may be stored up in the body for future use: but while stored, and when burned, they are only fuel, and become no more a part of our living body than does the coal in the bin, or in the firebox, become a part of the engine. The foods of the second group, the proteins, may become a part of our living protoplasm, just as new bolts, new pipes, new boiler plates, become a part of the engine. Not all

proteins do so, for proteid in excess of the immediate needs of the body for repair material is promptly burned up even though the body, just then, may not need fuel. There is, however, one difference which should be pointed out between our body and an engine; an engine never burns steel, but the body finally burns up for fuel all its worn-out protoplasm. Thus, sooner or later, all food is fuel. Some is used at once; some, the fat especially, which is stored up as reserve fuel, may not be used for years; and some is first made a part of our protoplasm, and later burned. In the animal body, proteins can be used only in the manufacture of protoplasm, which itself is a protein. But plants can manufacture protoplasm from the simplest substances—"bad air," water, and simple salts; this function is their great duty in the universe. Animals cannot do so, or can do so only in slight degree. The herbivorous animals must steal their protein (protoplasm) from the plants, while carnivorous animals in turn steal it secondhand from the herbivora, or thirdhand from weaker carnivora.

Such is the cycle of life; plants make, and animals appropriate. Of course during this metamorphosis the protoplasm does not remain alive, nor does it remain intact. The protein of broiled beefsteak, or of boiled peas, for illustration, is dead. Our body tears down these very complex, diverse, proteins into their constituent building materials, especially the amido acids which are relatively complex. This food material is carried around the body in the blood to the various organs. Out of these fragments, the cells of each of our various organs reconstruct for themselves the peculiar protoplasm which they need for their living structure. Fats and carbohydrates are never more than fuel. Water and salts, too, are practically unchanged while in the body.

Foods may be classified, also, as **nitrogenous and non-nitrogenous,** according as to whether, or not, they contain nitrogen, one of the main elements in the proteid molecule. Fats and carbohydrates contain no nitrogen. So important an element is nitrogen that, since it can be accurately esti-

mated, it is used as a measure of protein. For illustration, a beefsteak contains proteins, carbohydrates (glycogen), and fats. To find how much of it is protein, we find out how much nitrogen it contains; this figure multiplied by six gives the weight of the protein. So that, if we find in the total urine of one day 14 grammes of nitrogen, we know that the body has during the past twenty-four hours burned 84 grammes of protein.

The physiological problems of nutrition may be presented as four chapters, each presenting problems peculiar to itself. **In chapter one,** food is considered to be merely substance in the mouth, stomach, and bowel. In this condition it is **outside the body.** True, it is surrounded by the body, but it is not inside the body until it has been absorbed through the wall of the bowel. In chapter one, however, appear nearly all of the symptoms for which, in the past, diets have been prescribed, such as "dyspepsias," "indigestion," pains, nausea, vomiting, gas, acid stomach, diarrhœa, constipation. **In chapter two,** consideration is given to food that has been absorbed through the wall of the bowel, and is now **in the body and available for use.** In this chapter, we must consider the problems of malnutrition, diabetes mellitus, etc. **Chapter three** contains the story of that food which should promptly be used but which is not, and so is **stored up unused**; also the story of that food which should be stored up, but which for some reason cannot be properly warehoused and so is promptly oxidized, causing obesity, emaciation, etc., as some of the results. **Chapter four,** finally, deals with **the ashes of burned-up food,** which, of course, should be promptly removed. In this connection, arise the problems of nephritis, gout, etc.

The value of foods is calculated also according to the **amount of heat** which their oxidization in the body will produce. One gram of carbohydrates produces about four calories (4.1 cal) of heat (one **calory** is the amount of heat necessary to raise the temperature of one litre of water one degree centigrade); one gram of proteid produces also four calories

(4.1 cal), but one gram of fat furnishes about nine calories (9.3 cal) of heat.

The average daily diet of "the common man," that is of a man who weighs about 140 pounds, who works regularly, but does not do hard physical work, who is reasonably careful of his expenses, who considers himself well, and who pays no particular attention to the medical values of his food, has a heat value of from 2400 to 3000 calories. More accurately, such an average normal man of whatever weight consumes an amount of food which represents about 40 calories per each kilogram of his body weight. The man doing moderate muscular work would seem to need about 54 calories per kilogram, and the man engaged in hard muscular labor, about 70 calories per one kilogram of body weight.

Basal metabolism is a term which defines the heat production of the body when it is at rest, and at a sufficient time after the last meal (at least fourteen hours) to escape the stimulating effect of recently ingested food. For the above described average man, this amount of heat production is about 1600 calories a day (twenty-four hours) and for women about 1300. (On the relatively lower basal metabolism of women are based many theories, e.g. of her greater longevity, her ability to wear thinner clothes, etc.) It is this basal metabolism which we use in our diagnosis of diseases. It should be determined in the morning at about the patient's customary breakfast time, after a fast of at least fourteen hours and while the patient is lying quietly in his bed. If he has even stood up on his feet that morning, he should rest quietly in bed at least one hour before the test is made. The beginner is surprised to find what a big difference and so, possibly, what a bad error, even a slight excitement causing muscular tension, or restlessness, will make. In the case of a practically normal person who at that time was apprehensive and nervous we have seen the test even two hundred per cent too high. In that case the figures are, of course, not those of basal metabolism, and the test should be repeated each morning until the patient can co-operate satisfactorily.

Basal metabolism is now figured not in terms of body weight but of body surface, since the errors due to the various heights and weights of our patients are thus minimized. Thus, for the **average adult** the basal metabolism is **39.7 calories per hour per square meter of body surface.** This figure differs with sex, and slightly with ages. From this result, we calculate the basal diet which any given patient, whatever the sex, age or size, would, if normal, require, and what he, because of his present illness, does require. In conditions with **hyperthyroidism,** the basal metabolism is **always higher than normal;** in those of **hypothyroidism,** it is **lower than normal,** etc. **Fevers, cachexias,** etc., also **raise the basal metabolism.** When, as a result of therapy, the basal metabolism of a patient with one of these conditions shall have returned to normal, we know that we have been successful.

Balanced diet. The energy of our bodies **should not be furnished by one food.** Man could not live long on a diet consisting of proteid only, or fat only, or carbohydrates only. We must have sufficient proteid, but we must have also the other food stuffs. This means that we should **balance our diet wisely.** In order to do this, we determine, with the aid of basal metabolism, the necessary total caloric value of the diet, and then divide this among the foodstuffs, calculating each as grams per one kilo of body weight. The total basal diet is enough for the patient while confined to his bed. If he is an ambulatory case, not working, we increase this amount fifty per cent; and if he is working, we increase it still more, in proportion to the severity of his labor. In calculating the various foodstuffs, we consider that the adult needs per day between 0.75 and 1 Gm., the adolescent at least 1 Gm., and the child about 1.5 Gms. **of protein** per 1 kilo of body weight. Nevertheless, in the diets voluntarily chosen by healthy individuals who can afford good food, we find that the protein furnishes from 15 to 20 per cent of the total heat value of their diet, that is, about 2 Gms., or more of proteins per 1 K. of body weight. We cannot, however, approve of such a diet.

The great source of energy of our food are the **carbohydrates,** because of their economy, their abundance, and their ease of digestion and oxidization in the body. In general, these constitute about 60 per cent by weight of the total diet and furnish about 50 per cent of its total heat. The average person, therefore, cousumes per day between 3 and 5 Gms. of carbohydrates per 1 K. of body weight. **Fats** are much richer and more expensive foods than carbohydrates, but more difficult to digest. Of these, the average man consumes from 1 to 2 Gms. per day, and the rich gormand much more. **The average diet,** therefore, consists approximately, for a man who weighs 140 lbs.: of proteids 70 Gms., of carbohydrates 280 Gms., and of fats 140 Gms., all with a total food value of about 2735 calories. We believe, however, that the man described (if not engaged in arduous muscular work) would conserve his future health better on a diet of proteid 60 Gms., carbohydrates 300 Gms., and fats 100 Gms., total food value about 2400 calories. In disease, we must change the formula radically. For illustration, an average diet for a mild diabetic weighing 140 pounds would be, proteid 60 Gms. carbohydrates 123 Gms., and fats 185 Gms., while an epileptic's diet might well be, proteid 60 Gms. carbohydrates 20 Gms. and fats 232 Gms. The details of the various diets will be mentioned later in connection with the various diseases.

Vitamines. In planning diets, it is never enough merely to estimate calories and to balance the foodstuffs, etc., for we may feed a man the pure foodstuffs in proper amounts and in perfect proportion and yet he will die because of faulty diet. This is because **there are certain necessary foods called vitamines,** which also must be present, and the diseases which result, if one of these is withdrawn, are called deficiency diseases. Of vitamines we know at least five groups.

Vitamine A, or the fat-soluble-growth-promoting vitamine, is found mainly in the animal fats (with the exception of lard), and in the foliage of plants; therefore, in leafy vegetables, raw meats, milk, butter, and eggs. If the diet lacks

this vitamine, the child will be poorly nourished and stunted in growth. Medically we provide this vitamine best in cod-liver oil.

Vitamine B, or the water-soluble **antineuritic vitamine,** is present in a great variety of foods, but especially in the seeds of plants and the eggs of animals, therefore in eggs, milk, pears, beans, green vegetables in general, e.g., spinach and cabbage, and particularly in yeast. If the diet lacks in this vitamine, beri-beri may develop. Vitamine B is also essential for growth.

Vitamine C, or the **antiscorbutic vitamine,** is present in raw fruits, especially oranges and lemons, in vegetables, especially tomatoes, and in other "fresh foods." Cooking destroys this vitamine almost entirely, yet in the case of fruits and tomatoes the process of canning leaves it effective. A lack of it in the diet produces scurvy.

Vitamine D, or the **antirachitic vitamine,** is concerned chiefly in regulating the concentrations of phosphorus and calcium in the blood, and in controlling the calcification of the bones and teeth. This vitamine is present in cream, and especially in cod-liver oil, and egg yolk. Deprive the body of it and rickets will develop. Vitamine D can be produced in fatty substances through the action of ultraviolet light. The great benefit of ultraviolet radiation of the skin in rickets is thus explained, these rays activating the cholesterol of the skin.

Recently, other vitamines have been added to the list, especially **vitamine E,** which would seem to assist the body to proper sex maturity. This so-called **antisterility vitamine** is found in abundance in green leaves, such as lettuce, and in cereals, especially in the wheat germ.

It is important to take into consideration that since vitamines are produced by vegetables only (the activated lipoids of the skin produced by ultraviolet radiation of the patient's body being the only known exception) the vitamine value of, e.g., milk and its products will depend on the food which the cow has received.

CHAPTER IX

Diseases of the Upper Alimentary Tract

The œsophagus (Fig. 60) is a muscular tube, about nine inches long, through which the food passes from the pharynx to the stomach.

Sometimes the œsophagus contracts strongly, and for a time food cannot pass through it. Such **œsophageal spasm** occurs most commonly in nervous persons, who for long periods of time may, on this account, be unable to swallow solid food. The best known illustration of spasm of this organ, of course, is hydrophobia, since it is the feature which explains the name of that disease. **Real stricture of the œsophagus** preventing the swallowing of solid foods is, in over eight tenths of the cases which develop in adult life, due to cancer. These patients really starve to death, unless an artificial opening is made into the stomach, through which they can be fed. The removal of these cancers by surgery is seldom, if ever, successful. Among other common causes of the constriction is, in childhood, a scar following the burn by lye which the child has swallowed, and in young adults the constriction of the scar of a previous ulcer, especially that due to typhoid fever. In these cases the œsophagus may become greatly dilated above the stricture, that is diverticula, or pouches, may develop, and the food eaten will collect here until vomited. When vomitus is not acid to litmus such a condition may be suspected. The treatment for these benign constrictures is the dilatation of the structure by the passage of several dilators (sounds) each a little larger than the preceding. The diet of course should be liquid since even a slight organic stricture may because of spasm become temporarily complete when irritated by solid food. In addition to the above conditions the pressure against the œsophagus by any tumor low in the neck or in the chest, as a

goitre, aneurism of the aorta, etc., may have a somewhat similar effect as a stricture.

The gastrointestinal canal (Fig. 67) is a tube about twenty-five feet in length, through which the food passes, and in which the food is acted upon by various digestive fluids. By "digestion" we mean "liquefaction." The food which is in the stomach or bowel is not in the body; it is enclosed by the body, but is really outside it. A coin held in the mouth, for instance, is not more within the body than is a coin held in the closed fist. To get within the body food must, first, be rendered liquid, that is, soluble in water. When in solution, the food that is absorbed through the walls of the intestine, and enters the blood-vessels and lymphatics, is truly within the body. That part of the food which cannot be absorbed becomes later part of the fæces. Nearly all of the food we take is at first solid and insoluble. The process by which food is rendered liquid and soluble is digestion.

Of the foodstuffs ingested water is unchanged in the gastrointestinal canal, and is absorbed and excreted as such. **The inorganic salts,** also, are absorbed and eliminated practically unchanged. **The only carbohydrate** which our body can use is glucose, or the sugar of grapes, and this is present only sparingly in our food. Therefore, all carbohydrates eaten, if they are to be used, must first be changed into glucose. The carbohydrates of the food are for the most part in the form of starches, which are relatively insoluble. They are not, therefore, immediately useful to the body, although the riper, the softer, and the sweeter the fruits and vegetables are, when eaten, the more of their starches are in sugar form (for the plants store their carbohydrates in the form of starches, and, like our body, must transform them into sugar before they can use them). The number of carbohydrates which we eat is great. In the process of digestion all of them are first broken down to simple sugars, such as glucose, levulose, maltose, etc., and then all which are not glucose are "inverted," that is, are changed over into glucose by a change in the internal arrangement of their molecules.

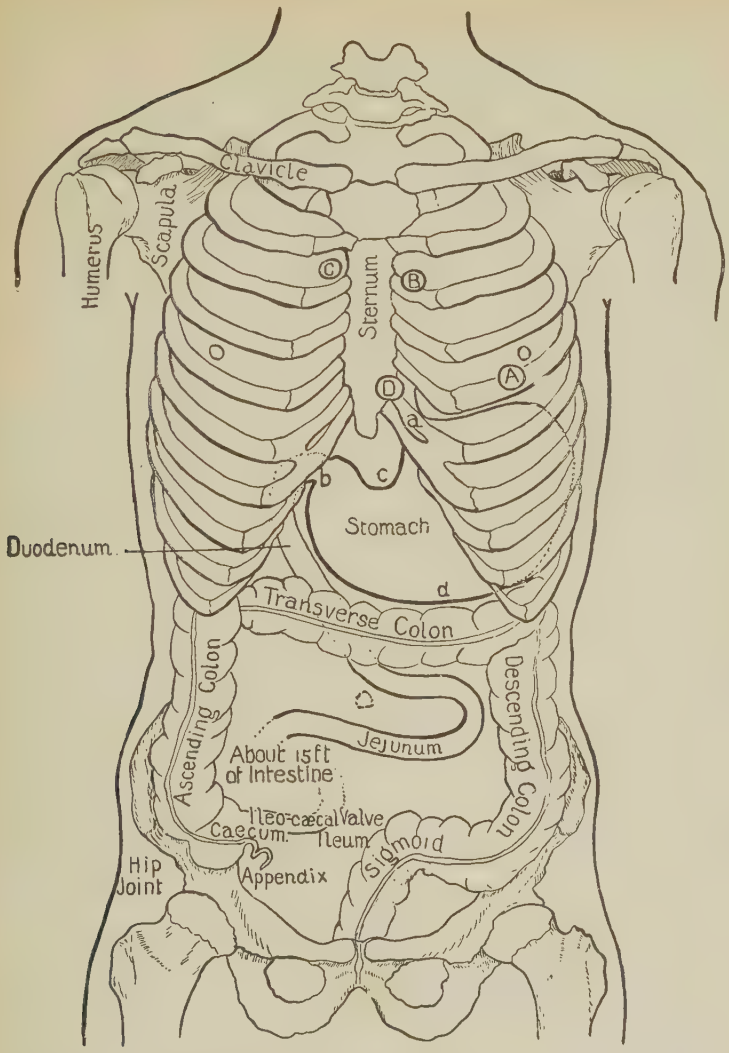


FIG. 67.—A diagram showing the position of certain organs. *A*, the point of maximum impulse of the heart, also called the "mitral area." *B*, the "pulmonic area." *C*, the "aortic area," and *D*, the "tricuspid area." The valves are not situated under these areas, but sounds made by these valves are best heard there. *a*, cardiac orifice; *b*, pylorus; *c*, lesser, and *d*, greater curvature of the stomach.

The fats are insoluble in water. All of them are composed of glycerine and fatty acids. Digestion can split this combination up into glycerine and fatty acids. The glycerine is easily absorbed, and the fatty acids unite with alkalies to form true soaps, which also are easily absorbed.

Proteins and albuminoids are very complex substances, and, in the forms eaten, are not soluble in water. By digestion they are split into many very much simpler substances, such as fatty acids, glucose, and especially **amido-acids** which are soluble in water and so are absorbed by the intestinal wall.

Once within the intestinal wall, some of these simple substances are **reconstructed,** the glycerine and soap are reunited into fats, and the simple products of protein digestion, circulating in the blood as amido-acids, are resynthesized to proteids by the tissues which use them. It is very much the same as though one were to tear down houses, and then were to use the bricks, timbers, and other building material to reconstruct other houses of different architecture. We eat a multitude of various proteins, since each animal and plant has proteins peculiar to itself. The bricks, timbers, **etc.,** (elementary substances) of which the various proteins of beef, chickens, vegetables, etc., are composed are much the same in all. They differ in relative amounts just as the differences in houses depend on the amount and arrangement of its building materials. In the body tissues all these various building materials are re-arranged into the few proteins of which our body is composed. The same is true of the fats of our foods.

In this chapter, we are interested in the first phase of the history of nutrition, that is, in the foods that are still outside the body. (See page 166.)

In the mouth, the food receives its first preparation for digestion, and starch digestion begins. **The solid foods are ground up by the teeth** into a pulp and **mixed with saliva,** which contains **ptyalin,** and this starts the starch digestion. The food may be in the mouth but for a few seconds only,

but this salivary digestion continues until the acid of the gastric juice has destroyed the ptyalin. The starches are, however, pretty well digested in the stomach. The reasons for this are: first, because it takes nearly thirty minutes from the beginning of the meal before there is enough acid present to destroy ptyalin; second, because it requires some time before the acid, even when abundant, can surround a bolus of food as swallowed and penetrate into this mass so as to stop starch digestion at its centre; but, more important still, is the fact that normally almost all of the acid is near the pylorus and scarcely reaches the food in the fundus of the stomach. Since the bulk of the meal remains at the fundus for at least one hour, most of the starch is well digested while in the stomach. We now see one reason why experience has taught us to plan our meals with the starches in the later courses, and one reason, also, why those conditions, such as gall-bladder trouble and appendicitis, which bring about the antiperistalsis which early carries the acid gastric juice back into the fundus, cause so much "indigestion."

There is much truth in the old German proverb that food "well chewed is half digested," for by **a few minutes spent in masticating the food we spare the digestive organs a great amount of work.** Gladstone considered that his good health was largely due to that fact that every mouthful of food was chewed 32 times, once for each tooth. A few years ago long chewing became quite a fad, and in honor of the man who has written a good deal about it, was called fletcherizing. The importance of the teeth in the maintenance of good health is illustrated in part by the improved health which old persons now enjoy, as compared with the health of those who were aged formerly, when false teeth were neither so well made nor so easily obtained as at present. Another reason why thorough mastication is desirable is because it stimulates the secretion of gastric juice.

Fluids pass quickly through the œsophagus and at once enter the stomach, but masses of solid food are held, by the

muscle ring at the cardiac orifice, just above the stomach for about six seconds. This ring then relaxes and allows the food to drop into the stomach.

The Stomach

Anatomy. The stomach (Fig. 68) lies between the lower end of the œsophagus and the duodenum. When empty, the normal stomach appears little larger than the duodenum; but

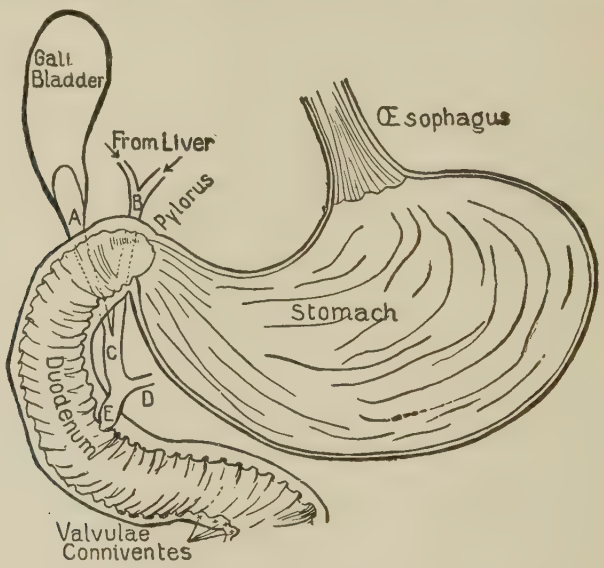


Fig. 68.—The normal stomach, duodenum, and gall ducts. *A*, the beginning of the cystic duct. *B*, the hepatic duct. *C*, the common duct. *D*, the pancreatic duct.

after a meal, it becomes a large, hollow receptacle, which holds about 1500 c.c. It lies in the upper abdomen, almost entirely on the left side of the body, and for the most part tucked away beneath the ribs. It has two orifices: **the cardiac orifice,** (Fig. 67, *a*) where the œsophagus enters, which lies from about 2 to 3 cm. to the left of the midline and deep in the abdomen behind the 6th or 7th costal cartilage, and which is provided with a circular muscle that can close it completely; and **the pylorus,** *b*, the communication between the stomach and the bowel, which lies more superficially, sometimes in the mid-

line just below the ensiform cartilage, and sometimes (when the stomach is full) 2 or 3 cm. to the right of this. **The lesser curvature,** *c,* or upper margin of the stomach, is from 3 to 6 inches long. The stomach at its widest point when full measures from 4 to 5 inches. The lower margin of the stomach, *d*, is called the **greater curvature.** This curvature in males crosses the mid line about one-half way between the ensiform cartilage and the navel; but in females, even in young girls who have worn no tight clothes, it always crosses the mid-line, after a meal, well below the navel.

Physiology. The first food enters an empty stomach. First, the pylorus closes tightly, and soon the walls of the stomach near the pylorus begin **to secrete gastric juice,** and at the same time in this same region gentle peristalsis begins, which mixes the food with the gastric juice. This peristalsis, however, does not early disturb the food in the fundus, but is most active over that part called the antrum pylori. The gastric juice contains **pepsin,** a ferment which splits protein, rendering it soluble; **hydrochloric acid,** which is necessary for peptic digestion; and **rennet,** which clots milk. Gastric juice is far from being a constant fluid, but differs according to the meal to be digested. For over an hour after a heavy meal, the bulk of the food remains in the fundus of the stomach, not yet exposed to the acid gastric juice, and so the ptyalin digestion proceeds uninterruptedly. Later, the peristalsis, or the churning motion of the stomach, will be more vigorous, and keep the food, now called chyme, at the pyloric end of the stomach, in constant motion, and constantly in contact with fresh gastric juice. As the mass of food is pressed towards the pylorus the portion which has already become fluid is allowed to enter the intestine, but that which is still solid is retained in the stomach until it is fluid. If at the end of a few (about seven) hours, any food is still solid, it is forced into the intestine by powerful expulsive contractions, more forcible than any which preceded. These are what make persons with indigestion wake up "with a sour stomach" soon after midnight.

Gastric digestion takes from one to seven hours. If at the end of seven hours after a meal, there is anything left in the stomach, something is wrong. During these few hours the protein has been about half digested, the fat has been separated from its mixture with other foodstuffs, and become partly emulsified, and over half of the starches are digested.

In the stomach practically no food is absorbed; all of this is passed on into the bowel, for it is not the function of the stomach to absorb digested food. Alcohol, however, and certain drugs can be absorbed through the stomach wall.

Gastric juice would seem not absolutely necessary. The stomach may secrete none, and yet, if its motility is good (and if no acid is present the stomach tries to empty itself with unusual rapidity) the patients have few symptoms. Also, a man can live even without a stomach. The question arises, therefore, what is the stomach for? In the first place it is a **reservoir in which is stored up the food eaten** occasionally and in large amounts. It is prepared here for the intestine, and is allowed to pass on into it frequently and in very small quantities at a time. Thus the bowel is supplied with food in just the amounts which it can handle. Remove a man's stomach, and he would have to eat very soft food, in very small amounts, and hence very frequently. He would spend the most of his time eating. With a stomach he can eat occasionally and large amounts. Also **the germs,** and especially the animal parasites in the food, **are practically all killed by the acid gastric juice.** In cases of long standing lack of acid in the stomach (achylia gastrica) these organisms thrive in the duodenum and upper jejunum and may prove very serious. And, finally, in the stomach **about half of the solid matter is digested,** that is, is changed to a liquid. The chief function of gastric digestion, however, and one perhaps more important than those already mentioned, is that it **prepares the food for the pancreatic digestion.** It produces changes in the food which the pancreatic juice cannot, and on which the success of pancreatic digestion depends. If achylia gastria is present, then the improperly digested foods

evidently cause serious disturbances in the small bowel. Indeed, the worst forms of anæmia are ascribed to this as cause.

Gastric juice will not be secreted simply because food lies in the stomach. Solid food might remain there for hours and not a drop of gastric juice be furnished to digest it. **The early secretion of this fluid** is in response to a **nervous stimulus.** For illustration, the odor, appearance, or taste of appetizing food stimulates, through the nervous system, the secretion of gastric juice. It is for this reason that we should enjoy the food we eat, and should chew it longer than we usually do. When we do not enjoy our food—either its appearance, or its odor, or its taste—there is not nearly so much gastric juice secreted as might be the case, although the food may be perfect in its nutritional qualities. This psychic stimulus produces what is known as a **primary secretion of gastric juice.**

The secretion of gastric juice is stimulated also by any food which is in **liquid form.** This soaks into the wall of the stomach and there chemically stimulates the secretion of gastric juice. This which is called **chemical or secondary secretion** is much the more important of the two. This is one of the chief values of soups, gravy, and sauces. It is caused chiefly, however, by the soluble products of the digestion due to the primary secretion of the gastric juice. Gastric secretion, therefore, digestion, depends much on the mind of the eater. A person who enjoys his food secretes more juice than a person who does not. If one is worried, or anxious, or frightened, there will be no nervous stimulus of secretion, and less gastric peristalsis to aid the digestion. The discovery of the importance of these two stimuli, the mental and the chemical, we owe, you will read in books, to a Russian by the name of Pawlow. But, as a matter of fact, this discovery was made centuries ago by some intelligent boarding-house keeper who was wise enough to serve soup as the first course. Suppose that at her table sat four men. Mr. A. enjoyed his food hugely, and so had a good

primary secretion. Also, he ate all the courses, including the soup, and so had plenty of chemical stimulus to secretion. Mr. B. enjoyed the solid food, chewed it well, but did not take soup. He probably had no indigestion, because the mental enjoyment of chewing the food furnished considerable gastric juice, and this soon digested enough of the solid food to cause also the chemical stimulus. Mr. C. did not enjoy his food at all, but did always take the soup. Although there was no mental stimulus to secretion, the soup was sufficient chemical stimulus; and so he soon had enough gastric juice, and therefore escaped dyspepsia. Mr. D., however, did not like his food and did not take soup. There was no mental stimulus to secretion, and, since he ate only the solid food, there was no chemical stimulus. His food rested undigested in his stomach, and he was sooner or later a confirmed dyspeptic.

Formerly, in all doubtful stomach cases, we examined the gastric juice. We do this more rarely now since the X-ray examination of the stomach is so satisfactory, giving us, as it does, accurate information concerning the motility of the stomach and often giving us visible evidence of an ulcer, cancer, or scar on its wall. Nevertheless, the **chemical examination of the gastric juice,** also, is sometimes very important. To get this, we wash out the patient's stomach the night before the test and the next morning give him a breakfast which consists merely of one shredded wheat biscuit and two glasses of water. Exactly an hour later as much of the gastric contents as possible is removed by a stomach pump. Lately, we have used a much smaller tube, the Reyfuss tube, which we can leave in the stomach for several hours and through which each fifteen minutes we remove a little of the contents for examination. It is doubtful, however, if this at present very popular method has much advantage over the former. The one very important point to be gained from this chemical analysis is an answer to the question **whether free hydrochloric acid is present,** or is all the acid which has been secreted bound? Just as soon as the

hydrochloric acid of the gastric juice comes in contact with proteid, or with any of its digestion products, it becomes bound to them; that is, it forms with them loose combinations which pepsin can digest. The normal stomach always secretes enough hydrochloric acid not only to unite with all the proteid bodies present, but it keeps on secreting until it has furnished a good excess of acid which, having nothing to bind with, remains **free.** The bound acid is necessary for digestion: the free sterilizes the stomach contents, controls gastric motility, etc. Since ordinary blue litmus paper is turned red by the bound as well as by the free acid we must use other color reagents, especially Congo red paper, which is turned to a very dark blue by free acid, but not by bound acid. It is very important in diagnosis that in **severe chronic gastritis, in gastric cancer, and in pernicious anæmia no free acid will be present.**

Thanks to the nervous system many persons suffer intensely from chronic dyspepsia whose stomachs are, so far as we can tell, without any trace of disease. The stomach is "all right," but it doesn't work right. Many persons refuse to believe that stomachs which pain them so severely are "all right." The man who is rolling about on the floor, screaming with pain, and begging for any operation which may cure him, is skeptical as to his stomach's being "all right." When we know that a stomach has not secreted any gastric juice for five years, it is hard to believe that this stomach is "all right"; the person who vomits so severely that he loses weight and strength, and thinks he is at the point of death, is slow to believe the trouble is not in his stomach; and yet such is often the case. In fact, of each ten (some say twenty) men with stomach symptoms, not more than one will be found to have stomach trouble. In one way the stomach is like a fire-bell. When the fire-bell rings, we do not rush to pour water on the fire-bell. No, we recognize that it is "ringing in an alarm" and after finding out where the fire is, we rush there. So in nine (some say nineteen) of each ten (or twenty) cases of "stomach trouble," if we

treat the stomach merely because it alone is troubling us, we would treat the wrong organ. The organ we should treat may be: the duodenum, if there is an ulcer there; the gall-bladder, when this is infected or contains gall-stones; the appendix, if it is chronically inflamed; the colon, in cases of chronic constipation; the nervous system; the eye, in cases of eye strain causing "bilious symptoms," etc. In this connection it is well to remember that there is no such disease as "acute indigestion." What we call that is, in nearly every case, an attack of gall-bladder trouble, often serious, often a gall-stone, or an acute appendicitis, or the pain of a gastric or duodenal ulcer. A beautiful illustration of an organic nervous disease which rings the "stomach alarm" are the gastric crises of locomotor ataxia. These patients have violent attacks of pain and vomiting, yet the stomach is perfectly normal; it is its nervous control which is disturbed. It behaves much as a steamship would were the officer on the bridge to give the engineer a succession of crazy orders.

Gastric Diseases

Nervous dyspepsia is a term which should be reserved for gastric symptoms which are certainly due to functional, not organic, nervous troubles, and by this we mean due purely to mental states. The result is a "gastric neurosis." In these cases the stomach is normal and the gastric symptoms appear only after emotional stresses and strains. In most of the neuroses the gastric symptoms are so small a part of the nervous condition that they attract little attention, but in some cases the general neurosis is so overshadowed by the severe stomach symptoms that we do not suspect the whole trouble to be what it is, a psychoneurosis. Such cases should be very carefully studied, however, since of course an hysterical person has the same opportunities to develop a duodenal ulcer or an infection of the gall-bladder as have persons less neurotic, and nothing is more embarrassing than to have a surgeon remove a large gall-stone from a person whom we had pronounced a case of nervous dyspepsia. In all cases of "acute indigestion" and of "nervous dyspep-

sia" all possible diagnostic methods should be employed before we finally decide on these diagnoses.

Among the motor neuroses is, first, supermotility. Such patients may have a condition known as **peristaltic unrest.** After a meal they can feel the peristalsis, and the stomach often makes loud rumblings which can be heard at some distance. **Aerophagia,** or "air swallowing," another motor trick, is not an unusual symptom in nervous persons. It may come on in attacks lasting hours. These persons belch large amounts of air with noise enough to be heard at some distance. One wonders where the air all comes from, but by watching them we can see that unconsciously they are continually swallowing it, and then, as it were, seeing how much noise they can make in belching it. **Nervous vomiting** also is a motor neurosis. Vomiting itself is never a gastric symptom alone; many muscles, controlled by the nervous system, do the most. There are some nervous patients who without reason vomit either occasionally or very frequently; some who vomit so often that they starve to death, and yet their entire trouble is a neurosis. Among these patients are some who vomit without nausea. They **regurgitate** their food and then spit it out. Sometimes this is a trick which a child learns and practices. We saw a very attractive young woman who had secretly practiced this when a young girl of fourteen years and had taught it to her friends. They even had contests to see who could vomit the most. As her friends grew older they outgrew this habit, but in the case of this patient the habit was not so easily broken, and gave her great concern when she was engaged to be married. "Rumination" or "chewing the cud"—that is, **regurgitating** the food from the stomach **and chewing it over again**—is a trick of some hysterical persons, but more commonly of those mentally deficient.

Cardiac spasm (cardiospasm) or painful contracture of the muscle which surrounds the orifice connecting the œsophagus with the stomach, is sometimes an hysterical symptom. In other cases it is caused by too hot or too cold food or by too

rapid eating. **Pyloric spasm** (pylorospasm) is a more common symptom. In this case the muscle closing the orifice between the stomach and bowel contracts vigorously and painfully at the time that the stomach wall is powerfully contracting in order to force the food on, and the result is pain as severe as is experienced in any true stomach disease. This may be a purely nervous symptom, but is more apt to be due to the presence, near the pylorus, of a small ulcer, which is irritated by the passage of food, or to gall-stones, or to appendicitis, which conditions reflexly stimulate the pylorus.

Secretory neuroses are not very common. The so-called "heart burn," hyperchlorhydria, **acid stomach, acid eructations**, etc., are not due to too much acid in the gastric juice. They are due to reverse peristalsis. Because of spasm of the pylorus, antiperistalsis churns the gastric juice back to the cardiac end of stomach where it does not belong, causing "heart burn," or forces it up the œsophagus into the mouth, causing "acid eructations." The patient is sure he has too much acid in his stomach because it feels so, but the trouble is that the normally acid gastric juice is where it shouldn't be. We repeatedly find normal persons who are unconscious that their gastric juice is much more acid than that of these patients.

Continuous secretion may be a nervous condition, although this is rare. By this we mean that the secretion of gastric juice continues when there is food in the stomach and when it is empty. In a great many of these cases, however, this is not a pure neurosis, no matter how nervous the patient may be, for the stomach on careful examination is found to be slightly enlarged and always to contain fragments of food which act as a constant stimulus to secretion.

Hypersecretion, on the contrary, may be a nervous condition. By this we mean the secretion, independent of food, of large amounts of gastric juice. These attacks begin suddenly, and then just as suddenly end. The best examples of hypersecretion due to organic disease are the gastric crises of locomotor ataxia, a serious disease of the nervous system,

but in which the stomach itself is not affected. A patient with one of these gastric crises suddenly has pain in the stomach and begins to vomit large amounts of clear gastric juice. Similar attacks of hypersecretion may occur as a pure neurosis, following immediately after sudden fright, or anything which excites or worries, as bad news. Almost immediately there is a gnawing pain in the stomach, the head begins to ache, and the man then begins to vomit incessantly, large amounts of clear gastric juice which is very burning and irritating, both in the stomach and in the mouth.

Subacidity, that is, the secretion of too little gastric juice (if no juice at all is secreted the term **anacidity** is used) is usually due to severe disease of the stomach, especially gastric cancer and pernicious anæmia, but it may possibly be a purely nervous condition. The patient is unconscious even of anacidity provided the motility of the stomach is good.

Gastric discomfort or even pain is usually present with all the motor neuroses already mentioned, but in addition to these some nervous persons have a **gastric neurosis which is chiefly sensory** in character. They may feel an intense discomfort during the digestion of each meal; there are others who are distressed by certain articles of food only, and that this is simply a nervous symptom is evident from the fact that if you prescribe one of these same foods as a medicine it will cause no pain, but let them eat it as a food and the pain is intense. The feeling in this case is of fulness, weight, and burning.

By gastralgia is meant severe paroxysms of gastric pain. We imagine that the most of these patients have a gall-stone, a duodenal ulcer, a kidney stone, or some other real trouble which causes reflexly a painful pylorospasm, and that others may be suffering from the gastric crises of early locomotor ataxia. A few, possibly, may have a truly "nervous" gastralgia, but it would be hard to prove this unless every diagnostic measure has been tried repeatedly. We can suspect it however if the patient is a very nervous person whose pains come only when events not connected with food upset them

nervously. Even then a surgeon may discover as cause a gall-stone or an ulcer, for these organic troubles may be very latent and cause pain only during the unusually active movements of the stomach which any intense emotion may cause even in the normal person; yet if we question these patients carefully we often learn that some of their attacks are independent of any disturbing incident.

The more we study the nervous dyspepsias the rarer do we find such cases. Some without doubt deserve this diagnosis, but the surgeons have taught us that the majority of cases of so-called "nervous dyspepsia" have a gastric ulcer, a duodenal ulcer, or the scar of an old ulcer, gall-stones, chronic appendicitis, latent cancer of the stomach, or even eye strain. The treatment of these cases will be found on page 555.

Acute gastritis is a complaint so common that we need not take much space to describe its symptoms. It is often due to some error in diet. The child eats too much or too rapidly, or eats food which is unsuitable because easily fermented or partly decomposed. The stomach will endure considerable of such abuse but sometimes becomes somewhat inflamed. But acute gastritis also is often the first sign of an acute fever, e.g., typhoid fever. The best example of acute gastritis, however, is that due to alcohol. The gastric mucous membrane of such a stomach when the seat of acute gastritis is red and swollen, secretes little gastric juice, and this contains very little acid but much mucus. The patient has uncomfortable feelings in his abdomen, with headache, lassitude, some nausea, often vomiting. This vomiting relieves him considerably, for it removes the irritating substance. The tongue is coated, and the flow of saliva is increased. If this irritating mass is not vomited, but reaches the bowel, colic and diarrhœa may be the result. As a rule the patient is well in about one day, although he may not have very much appetite for the next two or three days.

The treatment is to empty the stomach as soon as possible. To do this the patient can drink warm water, or mustard

and water, or stick his finger down his throat, and so induce vomiting. In addition one gives the patient castor oil or calomel in large doses, in order to empty the bowels as soon as possible, and this is wise even if the patient has diarrhœa.

Chronic gastritis is a diagnosis often made, but usually incorrectly. Most of the conditions thus diagnosticated are really cases of gall-bladder or appendix trouble. In fact, as we have said above, few of the patients who have stomach symptoms have stomach trouble. In true chronic gastritis the wall of the stomach is really diseased. Its mucous membrane is a little **thinned,** sometimes much—so much that there is scarcely any mucosa left; and **can secrete but little real gastric juice,** sometimes only water containing much mucus; and the **muscle wall,** as a result of the same process, is a little weakened and hence **less able to empty the stomach.** As a result it becomes dilated.

The most important **cause** of chronic gastritis is **infection** of the nose, throat and mouth. The pus swallowed is rich in bacteria which in time must injure the gastric mucosa. Among other causes are **foolish habits of eating.** The stomach is a long-suffering organ, and it is remarkable that it stands as much abuse as it does. Many persons overeat. Others eat food which has not been sufficiently masticated. Some eat large amounts of indigestible foods; others eat food good in quality, but improperly prepared. Some indulge for a long time in excessive amounts of fats or carbohydrates. Others overeat improper food, and in this connection should be mentioned hot breads, pies, cakes, ice water, soda water, tobacco (chewed), large amounts of tea, coffee, and alcohol. Irregular eating is also a common sin. It has been said that the platter kills more than the sword, and that overeating produces a greater number of invalids than overdrinking, the soda fountains more than the saloons. This refers of course to the number of such cases, it certainly has no reference to their severity. Then, **in certain general diseases chronic gastritis is an important feature,** as in anæmia, tuberculosis and Bright's disease. **Local conditions,** also, may bring about this

trouble, as the irritation of an ulcer in the stomach wall, or the stagnation of food in a stomach with muscle walls so weak that the food remains there an unusual length of time and ferments. And, lastly, chronic gastritis develops easily **in heart and liver troubles,** and in any other diseases which obstruct the circulation and allow the membrane of the stomach to remain turgid (chronically congested) with dammed-back blood.

The symptoms of chronic gastritis vary greatly. The appetite may be poor or too good (bulimia). There is usually some distress ("heart burn") after eating, or "gas," and some tenderness when we press over the upper abdomen. The tongue is coated; the taste in the mouth is bad; there is usually considerable nausea, and perhaps some vomiting, especially early in the morning—a vomitus which consists chiefly of mucus mixed with the products of decomposition of food; or, the person belches considerable gas. Constipation is the rule. The patient is usually low-spirited and complains of dizziness. If the gastric juice is examined chemically, there will be found little or no hydrochloric acid. In extreme cases there may be no gastric juice at all. Before we make a diagnosis of chronic gastritis we should prove by chemical analysis that the gastric juice is poor in quality, and by roentgenology that the motility of the stomach is weakened, since so often the above symptoms result from other conditions which cause pylorospasm (page 184) and will clear up after these shall have been relieved.

The treatment of chronic gastritis is to remove the cause. This may not cure the condition, because the stomach has already been injured, but it will prevent more injury and will lessen the symptoms. The person **should eat only proper food,** should chew it well, and, since the muscle of the stomach is weak, should eat small amounts at a time and hence more frequently than normally. These persons are usually very unwise in their choice of foods; they eat what they want. A list of the things allowed should be written out and the preparation of their food be supervised. It is often very wise to

put them on a pure milk diet for a short while at least. Grains, vegetables, and the fats should be avoided; the hot breads, the pastry, and the fried foods should be omitted. To assist the stomach to digest the food, the patient can take **hydrochloric acid or pepsin, or both.** Dilute hydrochloric acid, from 15 to 25 drops at repeated intervals after a meal, is far more important than pepsin, since it is the acid which disappears first. Usually there is enough of the ferments present, but they cannot act because there is a lack of the acid. It is often desirable **to wash out the stomach each night** to keep it clean, and we may use water, or, better, a 1 per cent salt solution, or a 3 to 5 per cent soda solution. The most valuable drug in such a case is really some **bitter tonic,** such as gentian, nux vomica, quassia, etc., for these stimulate the secretion of gastric juice. The patient often gets relief by drinking large amounts of alkaline water at night or early in the morning, and some find it an advantage to take large amounts of salt. Above all else the psychic side is important, and whatever the patient eats he should enjoy. **Some,** at least, **of his food should be liquid** in order to obtain as much chemical stimulation of secretion as is possible. Among other drugs may be mentioned bismuth, small doses of chloroform, and the compound spirits of ether.

The dilated stomach may hold even from 3 to 5 quarts, while the normal stomach has a capacity of about 1500 c.c., or about 3 pints. A patient with a stomach which for months does not secrete any gastric juice may be entirely unconscious of any trouble, provided the stomach empties itself with normal ease; but one with a stomach very slightly enlarged is soon painfully conscious of it, so important is its complete emptying. If food remains in it even a little too long there will be symptoms, because of the decomposition or the weight of the food, or the effort of the stomach to empty itself.

Acute dilatation of the stomach occurs especially after surgical operations and in acute fevers, such as pneumonia or typhoid fever. The abdomen swells, the vomiting is severe,

and, unless the stomach is promptly washed out, the danger is great.

Chronic dilatation is a very common condition. By this we mean not only a large but a weak organ. A stomach may be very large, but we would not on this account call it dilated provided it empties itself within the normal time. Such, we would call a case of megalogastrica. Also, the stom-

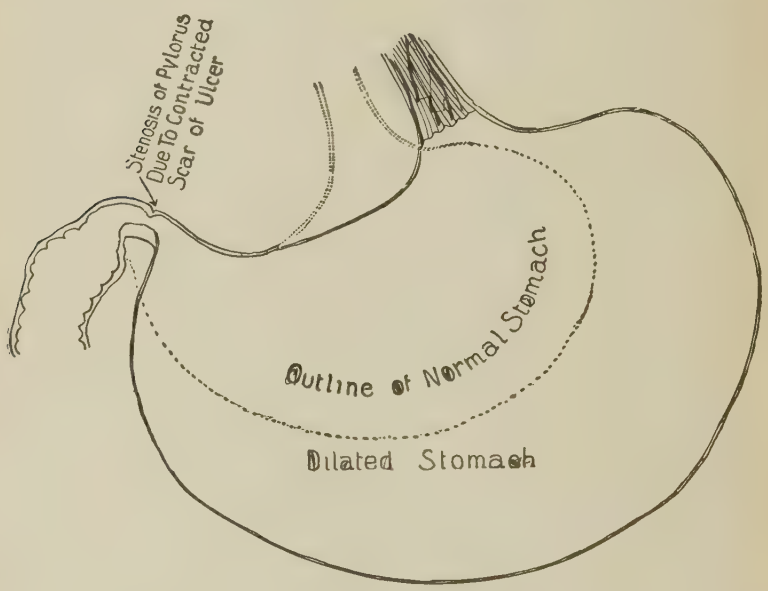


Fig. 69.—Dilatation of the stomach due to the contracted scar of an ulcer.

ach may seem large and yet not be dilated. The best illustration of this is a condition called **gastroptosis** or falling of the stomach, which occurs especially in nervous women. Their stomach has descended to a much lower position than normal, the greater curvature has descended even into the pelvis, and if the stomach does not empty itself in normal time (which it may not) symptoms result. Sometimes not only the stomach but the liver, kidneys and other organs also have "dropped down," a condition called **visceroptosis.**

Chronic dilatation results, first of all, from some **obstruction at the pylorus.** There may have been there an ulcer which has healed, and the scar of which contracted. Sometimes this so constricts the pylorus that a lead pencil can hardly be pushed through the opening through which one ought easily to put three fingers. In other cases the repeated excessive contractions of the pylorus have made its muscle bands to hypertrophy so that it almost fills the aperture. A more common cause is a **tumor** which grows at this point. This tumor may be small, it may be as large as a fist; but when well placed it may be small and yet very effectively stop up the pylorus. In other cases a tumor outside the stomach closes the pylorus by pressing against it. Other patients have had an **inflammation near the pylorus**—in the gall bladder, for illustration, which produced "adhesions" which when contracting may almost close the pylorus, or so kink it that the food can hardly pass through. In all these cases the trouble is at or near the pylorus, but not in the stomach wall. Since, because of the obstruction, the muscle of the wall must work unnaturally hard to force food out of the stomach, the result is what one would expect; that is, it hypertrophies, even to from 2 to 3 times the normal thickness. If we watch the abdomen of such a patient two or three hours after a heavy meal, we can usually see the commotion which the vigorous contractions of the stomach wall cause.

The second cause of chronic dilatation of the stomach is **weakness in its own wall.** The pylorus is normal, but the muscle is too weak to force the food through it properly. This weakness may be the result of habitually overfilling the stomach, or of the chronic inflammation of chronic gastritis; or it may be a part of a general weakness of the whole body, such as occurs in anæmia, tuberculosis, cancer, or rickets.

So long as the weakening muscle is able to empty the stomach there may be no symptoms at all, but often there are nervous symptoms, and distress after eating. Sooner or later the stomach doesn't quite empty itself, and **vomiting**

begins to expel that portion of the meals which does not reach the duodenum. The patient may vomit only a small amount of food, but if the condition is of so extreme grade that little passes on he may vomit 3 or 4 litres, and in this vomitus can be recognized, usually in putrid condition, the food eaten two or even three meals before, or even three or four days before. The food ferments or decomposes; it "sours" in the stomach. This stagnant food so injures the stomach wall that soon it secretes a gastric juice poor in quality and quantity. Since so much food is lost as vomitus, the patient loses weight and strength.

The diagnosis may be made from the vomitus, because if this **contains food eaten over seven hours before,** the stomach is quite certainly dilated. If the person be made to drink a teaspoonful of soda dissolved in an ounce of water, and then, a minute later, a teaspoonful of tartaric acid in the same amount of water, the carbonic acid formed will inflate the stomach and we can study its size. Sometimes it fills almost the whole abdomen.

For treatment, the person should improve his habits of eating. Since the stomach has greater difficulty with a large than with a small meal, he should each day eat five small meals rather than three large meals. **The diet** should be food easily digested: milk, eggs, scraped beef, etc., but no coarse vegetables. If the dilatation is severe the stomach should be **kept empty by washing it out each evening,** for if allowed to remain dilated all the time it loses more of its tone than the disease alone can account for. But if washed out regularly it may recover much of its strength, provided there is a not too great obstruction at the pylorus. The person should eat a pretty dry diet, and avoid such foods as ferment easily; such as fats, starches, and vegetables. For medicines, **the bitter tonics** are to be recommended. For extreme cases surgical treatment is the only resource, and the pylorus can, by a simple operation, be restored to its original size.

By ulcer of the stomach we mean an excavation formed in the gastric wall by the death of a small area of the mucous

membrane. This hole may reach down to the muscle, or even through to the peritoneum.

It is a question, which formerly was much discussed, why the stomach does not digest itself. Surely, the fact that it is living tissue does not protect it, for other live tissues if placed in the stomach are readily digested. We cannot here discuss this question concerning living mucosa, but there is no doubt that if a portion of the wall is poorly supplied with

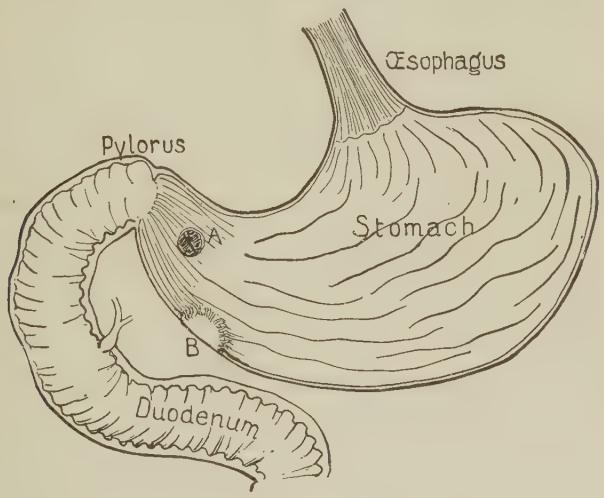


FIG. 70.—Ulcers of the stomach. *A*, a small punched-out ulcer near pylorus. *B*, a larger ulcer cut across.

blood, or dies for any reason whatever, that portion is quickly digested and an ulcer is the result.

Small superficial ulcers of the stomach are probably very common, do no harm, and rapidly heal over. The ulcers which make trouble seem to be those which are so located that continuous motion of the muscle wall prevents healing—for instance, near the pylorus, that part of the stomach which moves most in peristalsis.

Gastric ulcers are common enough, but it is strange that they should be more common in certain localities, as Great Britain and Massachusetts, than in others. Ulcers develop

with greatest frequency between the ages of 15 and 25 years, and especially in anæmic persons. They also occur in those persons whose work causes constant pressure on the stomach, such as the workman who always leans over the edge of a desk; the shoemaker, who presses the last against the abdomen; the persons who constrict their waists too much by tight clothing.

As a rule ulcers are single, yet there may be many, even from 5 to 30 at one time. Nearly all of them **occur near the pylorus,** for the reason, perhaps, that in this position they would have the greatest difficulty in healing. Those just beyond the pylorus, the duodenal ulcers, usually are described with the gastric ulcers because their symptoms are similar, but the differences in the general treatment of the cases are important enough to require separate description (page 200). Ulcers may be small with clean edges, as if punched out by a sharp instrument, or they may be as large as the palm of the hand. Those larger than a quarter, however, may not be (some say never are) simple ulcers but very sluggishly growing cancers. Some have terraced edges. When they heal they **leave, as a rule, a scar,** and this scar contracts with results which may be very serious if the ulcer was large; for, near the pylorus (Fig. 69) it can almost or quite shut off this aperture, and if near the middle of the stomach it can so **constrict this zone** that the so-called "hour glass" shape results. An ulcer may completely **perforate the stomach wall.** If it does this slowly the chances are that before there is an actual perforation the stomach will have become adherent to a neighboring organ, to the diaphragm or the liver, for illustration—and therefore little damage will result; but, if this has not happened, the stomach contents will pour through the perforation into the peritoneal cavity, and either a local abscess or, unless an emergency operation is performed, general peritonitis will be the result. The small arteries in the floor of an ulcer usually remain patent, and since their exposed wall becomes weakened, a little aneurism about the

size of a small bead forms, which often ruptures and gives rise to the **hæmorrhage** which is so common in these cases.

The symptoms of gastric ulcer are varied. There are three called "classic" since they are so common. While the patient may have a large ulcer and yet never know it, probably because the ulcer is not near the pylorus, yet as a rule he will complain of "dyspepsia," that is, of **discomfort** about one to three hours after a meal, but **beginning,** with remarkable constancy, **at the same time after each meal.** This disappears, however, when the stomach is empty, so that both the first hour after a meal and the one preceding the next meal are comfortable ones, the stomach, therefore, giving the so-called quadruple rhythm; food, comfort, pain, comfort (see page 201). This discomfort from 1 to 3 hours after a large meal often amounts to actual pain, which may be so great that the patient will roll in agony on the floor; or, when less severe, he may relieve it by pressure, and so he leans over the back of a chair or lies across a hard pillow pressed against the stomach. There is usually **a sore point** which the patient can point to, just below the ensiform, or to the left of the midline, and often another in the back in the corresponding position, where even slight pressure is painful and even the skin sensitive. This pain after eating is probably explained by the irritation of the ulcer by the food. When one remembers that in churning the food the normal stomach uses considerable force, he wonders that the pain is not greater than it is.

The second symptom, which not half the cases of gastric ulcer have, is **hæmorrhage.** A few of these cases suddenly vomit a large amount of blood—a pint, or a quart, or even more. In the majority of cases with hæmorrhage, the stools only will contain much of the blood and as a result will be tar-like (the result of digestion) because the blood will flow on into the intestine. The patient often becomes very anæmic, almost exsanguinated, and an extreme secondary anæmia may result.

The third classical symptom of gastric ulcer is **vomiting.** It is often induced by sticking the finger down the throat,

but, at the height of the pain, it may come spontaneously. In its lesser degrees the vomiting is replaced by symptoms called hyperacidity. By this we do not mean that the patient actually has a too acid gastric juice, for he seldom, if ever, does; but he has symptoms which suggest it, heartburn, eructations of acid fluid, acid vomitus, etc. The explanation is the antiperistalsis described on page 184.

In the recognition of gastric ulcers the X-rays prove their value, since the great majority can be thus demonstrated.

The treatment of gastric ulcer is, of course, to allow the ulcer to heal. This may be done by complete rest in bed, and by a diet and medicines which will not allow any acid to remain free in the stomach. Sippy's treatment is the best. The patient is given, every hour on the hour, three ounces of a mixture of half cream and half milk, and every hour on the half hour a dose of ten grains of sodium bicarbonate and ten of magnesium oxide, or twenty grains of sodium bicarbonate and ten of bismuth subnitrate. Later, any soft food rich in fat may be given. For years after he is cured the patient should eat at regular hours meals of simple and fatty foods, and should take considerable alkali after meals to keep the stomach contents as alkaline as possible. Of all the meats, veal is most easily and quickly broken up into a soft mass, and hence is especially to be recommended. If the patient has had a severe hæmorrhage, the anæmia, of course, will demand attention. In case there is suspicion of perforation, the surgeons should operate at once and remove the ulcer. X-ray treatment over the stomach, which allows the ulcer to heal by reducing the gastric acidity, is now a popular treatment.

By cancer of the stomach we mean, first, a new growth, a tumor. Just as a wart which forms on the skin is something which is not normally there, but is an enlargement of a tissue which is there, so cancers of the stomach are new growths of the gastric mucosa. But cancers differ from other tumors in that the latter, e.g., the warts, cause trouble only where they grow, while cancers grow "wild," that is, invade the neigh-

boring tissues. Fragments of cancer, also, are carried by blood and lymph stream throughout the body, and wherever they settle they are the starting points of new cancers, which are called "metastases." Cancers, also, often slough very easily, so that what was a hard mass is soon an ulcer; but in the wall of the ulcer is the spreading cancer. Cancer is a disease of middle age. It is rare to find one in a person under 40 years of age, and yet they do occur.

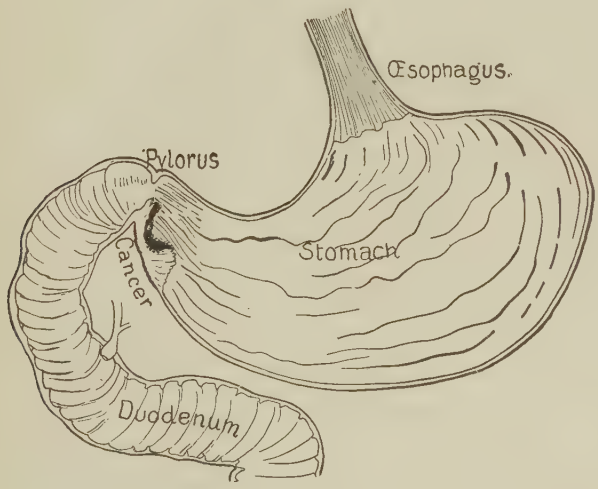


FIG. 71.—Cancer of the stomach at the pylorus.

The symptoms of cancer of the stomach are numerous. First, there are certain symptoms which are **common to cancers anywhere** in the body—that is, loss of weight and of strength, and a progressive anæmia. These symptoms are, however, most marked in cancers of the gastrointestinal canal, and may be absent in the very acute cases. Then, we have **special symptoms** due to the presence of the cancer in the stomach, and the most important of these are **sudden loss of appetite, especially for meat,** and a **dyspepsia of not over two years' duration.** If two persons were to describe their dyspepsia in just the same words, but one were to say that

his trouble began when he was a young man and had been present more or less ever since, while the other were to declare that he had had a normal digestion until he was forty years old or more, we might be reasonably sure that the former had no cancer, while possibly the other has. If, in addition to this dyspepsia, developing suddenly at middle age, he has also lost weight and strength, and has become rather pale, we may be more confident of our suspected diagnosis.

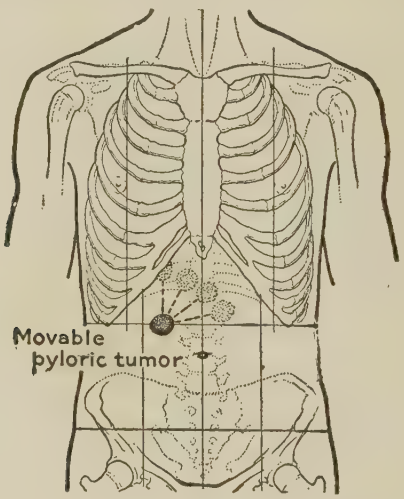


FIG. 72.—Movable cancer of pylorus. (Osler.)

Then, there is the vomiting of **coffee-ground vomitus.** The blood which leaks slowly from the cancer (large hæmorrhages are very rare in cases of cancer) becomes partially digested and therefore of a dark coffee color. The patient may not vomit, but we can constantly find **traces of blood in the stools** if we examine chemically for it. This is a very early valuable sign of a cancer anywhere along the gastro-intestinal canal. Also the patient may complain of actual pain in the stomach.

If we examine the gastric juice, we shall find that very early there is **no hydrochloric acid present,** but that **some lac-**

tic acid is present, formed from the gastric contents. To be of value as a sign, this lactic acid must be formed within one hour after the meal, and of this we can be certain only if we have carefully washed out the stomach before that meal. We may be able to feel the **tumor** of the stomach. We may find in the abdomen other tumors also, metastases of this gastric tumor, especially on the surface of the liver or in the skin at the umbilicus, etc.

The third group of symptoms of cancer of the stomach includes those **which depend on the exact location of the cancer** in the stomach wall. If it is at the cardiac orifice, vomiting will immediately follow the meal. If it is near the pylorus, it will cause an obstruction there, and then all the symptoms described under dilated stomach and, later, of chronic gastritis will also be present. Lastly, we have a certain number of **patients who have no symptoms at all,** but, dying for some unknown reason, are found at autopsy to have a cancer in the stomach. In the early diagnosis of gastric cancer X-ray examination is very valuable, demonstrating the trouble long before the tumor can be felt.

The early treatment of cancers of the stomach is surgical. If the operation is early enough the disease may be entirely removed and the patient get entirely well. Even if it is a little too late, an operation may add several years, and these years of comfort, to the patient's life, for most of the distress that a patient with cancer has is due to his dilated stomach. Even when the cancer is so far advanced that no attempt should be made to disturb it, if there is pyloric obstruction the intestine is sewed to the stomach and a new pylorus made (posterior gastroenterostomy). As a result, a patient with even a large cancer may be made comfortable by proper diet, by keeping the stomach well washed out, etc.

CHAPTER X

Diseases of the Intestine

Anatomy

The intestine is a muscular tube which begins at the pylorus (Fig. 67, *b*) and ends at the anus. The first twelve inches from the pylorus are called the **duodenum** (Figs. 67, 68, and 70); into this part enter the gall duct (Fig. 68, *C*) and the pancreatic duct (Fig. 68, *D*). The small intestine is about twenty feet in length. The upper two fifths below the duodenum are the **jejunum,** the lower three fifths are the **ileum.** The ileum opens into the colon, or large bowel (Figs. 67 and 78). Just below the juncture of the ileum and colon—a point which is protected by a valve, the ileo-cæcal valve, opens the **appendix vermiformis.** The large bowel is about five feet long. It begins as a pouch, the **cæcum** (Fig. 78), into the side of which the ileum and appendix enter, and first ascends to the liver, as the **ascending colon,** then crosses the abdomen, as the **transverse colon;** it then descends, as the **descending colon,** into the left flank. The next part is named the **sigmoid flexure,** and this passes into the **rectum.**

In the stomach the food is gradually reduced to a very soft pulp. The stomach begins digestion but does not absorb the digested food—that is one of the chief functions of the intestine.

The Duodenum

Duodenal ulcers are almost twice as common as those of the stomach. While roentgenological examination alone can determine with certainty on which side of the pylorus some of these "gastric" ulcers lie, yet certain symptoms are very suggestive of their location in the duodenum. In the first place, **the average age of patients** with duodenal ulcers is **older** than that of those with gastric ulcer; there is **more often evidence of gall-bladder disease also,** which indeed may be an indirect yet important cause of the duodenal ulcer;

it is the duodenal ulcers which cause **regularly recurring hunger pains** which are relieved promptly by eating; the gastric **distress after eating comes later** after meals than in cases of gastric ulcer and continues as hunger pains, giving rise to the triple rhythm of "food, comfort, pain" (see page 195); **vomiting is much less frequent**; and **seldom is there bloody vomitus,** but more often large, tarry stools; the **tenderness on pressure is lower** than that due to gastric ulcer, and usually is just above and to the right of the umbilicus.

Into the duodenum, at a point eight or ten centimeters below the pylorus, open the **pancreatic duct and the common bile duct. The pancreatic juice** (page 251) contains three ferments—trypsin, diastase and steapsin. **Trypsin** is the most powerful ferment known. Its action is similar to that of pepsin, in that it breaks down the protein molecule to simpler and simpler bodies, but it is infinitely more powerful than pepsin, breaking down the protein much more completely, and with much greater rapidity. So powerful is trypsin that there is little wonder that the body protects itself by manufacturing it as trypsinogen. That is, the pancreatic juice as it flows through the pancreatic duct cannot digest protein at all; but let it come into contact with the mucous membrane of the intestine, and harmless trypsinogen is changed at once into powerful trypsin. This is due to enterokinase, a ferment in this mucosa which "activates" the trypsinogen. Another difference between trypsin and pepsin is that while pepsin can act only in an acid juice, trypsin requires an alkaline medium. Nevertheless, trypsin cannot split some proteids at all unless they have already been acted on by pepsin.

Diastase, or amylopsin, is practically the same as the ptyalin of the saliva. That is, it breaks down the starches and the complicated sugars to the simple sugars—glucose, levulose, etc.

Steapsin, or lipase, is a ferment which splits fat into its two components, glycerin and fatty acids. The pancreatic juice sometimes contains other ferments. In children it contains lactase, which is important in digesting milk sugar. The

pancreatic juice is also very alkaline and so changes the acid food from the stomach into an alkaline food and furnishes some of the alkali which, combined with the fatty acids, makes soap.

The bile also enters the intestine at this point (Fig. 68, *c*). Many of the constituents of bile are substances which are no longer of use to the body. It contains also alkalies, which help to neutralize the acid from the stomach, and bile acids in which soaps and fats are soluble, and which aid much in the absorption of fat in the intestine.

Along the whole length of the small intestine the mucous membrane secretes small amounts of an **intestinal juice** which may help a little in digestion. The mucous membrane of the small bowel, however, does contain **important ferments** which evidently act on the food during its absorption. One of these, erepsin, completes the digestion of the protein; another helps split the fat; while the "inverting" ferments are important in carbohydrate digestion. While ptyalin and amylopsin split the complex carbohydrates into simple sugars, these ferments "invert," or change, all the simple sugars into glucose.

In review, then, water and salts are not digested. **Water** does not stay more than one or two minutes in the empty stomach, but is passed on into the bowels and is there absorbed. **The salts** also are absorbed there. The digestion of carbohydrate begins in the mouth, proceeds in the stomach until the ptyalin has come into contact with the acid of the gastric juice (page 175), and is continued by the pancreatic juice. **All the complex carbohydrates**—starches, sugars, etc., —are first broken up into simple sugars, and these, the inverting transforms to glucose, the only form of sugar of much use to the body. **Fats** are split in the stomach to a limited degree, but for the most part this is done by the pancreatic juice, which also furnishes the alkali that changes the free fatty acid into soap. The soap and the glycerin are then absorbed. **The digestion of protein** begins in the stomach. The pepsin breaks up the higher proteins into simpler bodies, such as peptone, etc. Trypsin quickly breaks these up into much

simpler bodies, and erepsin finishes this process. The process by which all of these digestion processes are done is called "hydrolysis"; that is, a molecule of water is the wedge and a ferment the mallet by which these huge molecules are split into smaller ones. There are some proteins which pepsin cannot attack at all. There are also other proteins which trypsin cannot digest unless pepsin has already begun the splitting. There are still others which trypsin can digest, but it does so better when pepsin has already begun the process. Thus we can say that gastric and pancreatic digestion work together. The pepsin prepares the way for the trypsin.

Bile, although formed almost continuously, does not flow into the intestine except during the time of digestion. The rest is stored up in concentrated (even from five to ten times) form in the gall-bladder, which serves as a reservoir. It probably is this stored-up bile which enters the duodenum first when intestinal digestion begins. **Pancreatic juice** is not secreted while a person is fasting. When, however, food reaches the stomach, then the flow of pancreatic juice begins. It reaches its maximum about three hours after the meal, the time when most of the food is being poured into the duodenum, and then slowly diminishes, as the food is gradually digested. It is of interest that the stimulation for pancreatic secretion is chiefly chemical. A substance called **secretin** is formed in the stomach and intestine, is absorbed in the blood, is carried to the pancreas, and there stimulates this organ to secrete pancreatic juice. We can, therefore, make the pancreas work at any time we wish by injecting into the blood of an animal or person a little secretin from another animal.

The composition of **gastric juice is not always the same.** In fact, the gastric juice varies according to the task before it. So it is with the pancreas. The amount, the quality, and the quantity of the pancreatic juice intended for each meal depend in some degree on the food there to digest. The trouble some have to digest milk may be an illustration of this. In young animals on a milk diet, the pancreatic juice

contains lactase, a ferment quite important in the digestion of milk. Give them no milk for a few days and this lactase disappears. After a person has ceased to drink milk, lactase is thought to disappear, and so when he begins to drink it again, at first it distresses him. Since it is very necessary that some patients should drink large amounts of milk, we start by giving it to them in small amounts. At the end of about a week they easily digest five or six quarts of milk a day, and

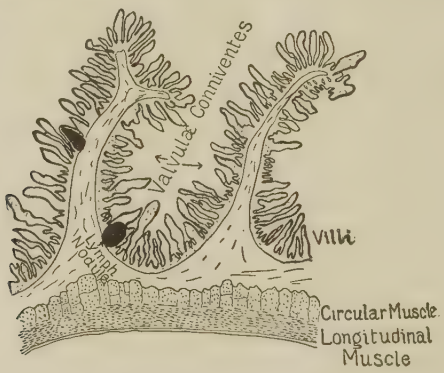


FIG. 73.—A section through the wall of the small intestine (jejunum) made parallel to the length of the bowel, showing the folds of the musoca, and the villi on the folds. (Magnified.)

without a single symptom. So it is with all our meals. The gastric and pancreatic juices are modified to meet the requirements of each meal.

THE JEJUNUM AND ILEUM

The intestinal wall contains four or five ferments. **Enterokinase** has already been mentioned as the one which activates the trypsinogen; **secretin,** as the substance which stimulates the pancreas to secrete its juice; erepsin, which completes the trypsin digestion; and an **inverting enzyme for each sugar**—maltase to invert maltose into dextrose, invertase, to invert cane sugar into dextrose and levulose, and lactase to change milk sugar into dextrose.

The function of the small intestine is to complete digestion and absorb the food, which is now in fluid condition. The

mucous membrane lining the wall of the small bowel is thrown into folds to increase its surface (Fig. 73), and not only that, but has a surface much like velvet, being completely covered with finger-like processes, known as villi (see Fig. 73), which vastly increase its absorbing surface.

By the time the food reaches the ileo-cæcal valve, practically all that is of value has been absorbed. That which is left is the indigestible part of the food, especially cellulose, together with some of the constituents of the bile and intestinal fluids, also, a great many old epithelial cells (for the mucous membrane of the intestine, like the external skin, is constantly throwing off its used-up cells), and, finally, vast numbers of the bacteria which grow in the intestine.

The Colon

After a heavy meal the first of this refuse reaches the ileo-cæcal valve in from two to five hours, and the last nine hours later. **In the large intestine** this fæcal matter very slowly moves towards the rectum and should be evacuated in from twenty-four to forty-eight hours. It enters the colon mixed with much fluid. Here **the water is absorbed,** and the intestinal contents are gradually dried down to a solid mass. **In the small intestine are many bacteria,** chiefly the milk sourers (Bacillus lactisærogenes), but in the large intestine Bacillus coli communis, usually called the "colon bacillus," predominates. When evacuated, perhaps one fifth or more of the solid (and dried) stool is made up of the bodies of bacteria. The function of these bacteria nobody knows. Many think that they are a benefit to us. In the large intestine they seem to help destroy whatever food may have escaped digestion.

By diarrhœa one means the passage of too frequent and too soft stools. It is not a disease in itself but a **symptom of many conditions,** some temporary and trivial, some very serious. When the diarrhœa is a symptom of a definite inflammatory disease of the large bowel we consider it a **dysentery** (page 207), and reserve the term diarrhœa for mild,

temporary, or purely functional conditions. As has been said above, the intestinal contents enter the large intestine as a fluid, and the function of the large intestine is to absorb the water. The stools will therefore be abnormally soft if the **peristalsis of the large intestine is so rapid** that there is not time to absorb the water, or if disease of the bowel wall prevents this absorption, or if fluid is actually added from the blood to the intestinal contents. The latter occurs in most acute bacterial infections of the intestine, but the most marked illustration is Asiatic cholera, a disease in which the fluid of the stools comes for the most part from the blood (see page 513). **Diarrhœa may be caused by food.** The patient may have eaten too much food, or not chewed it enough, or the food may be unsuitable, as unripe fruit, which cannot be digested, or over-ripe fruit, or spoiled meats, which decompose rapidly. In these cases the food is an irritant and causes irritation and increased peristalsis of the bowels. Popularly this is called ptomaine poisoning. Seldom, however, is any true ptomaine (alkaloids derived from foods) present, and the great majority of these cases should be called **food infection.** Some, however, are really **acute gall-bladder attacks** (page 235), excited by the infection from the food. Conditions like these, while called diarrhœa, are really a mild enteritis, but one which clears up promptly as soon as the irritating substances have passed. There are **some perfectly good foods** (eggs, shell fish, etc.) which are poisonous to certain persons and always give them diarrhœa. Some **disturbances of internal secretions** cause diarrhœa, as in exophthalmic goitre. **Achylia gastrica** causes diarrhœa, probably since the food enters the bowel improperly prepared. **In achylia pancreatica** (lack of pancreatic juice) there is a marked diarrhœa because the food is not properly digested. Also, **increased peristalsis** may be caused by true poisons, such as arsenic, mercury, etc. **Certain infectious diseases** may be accompanied by diarrhœa, as typhoid fever and tuberculosis. Here it is probably the actual disease of the bowel wall that explains the diarrhœa. In **heart and liver diseases**, with chronic pas-

sive congestion of the bowel wall, (see page 120) there is often diarrhœa; while other persons, when merely run down, have frequent, loose stools.

Changes in the weather will give some persons diarrhœa, especially if the weather turns suddenly cold.

In many cases, however, the trouble is not in the food or the intestine, but in the mind of the person; for the rapidity of peristalsis of the colon is in some measure controlled by the nervous system. Some persons have **a nervous diarrhœa**; that is, a diarrhœa the result of abnormal nervous control of the bowel. Such persons may have after each breakfast one or two loose movements. They are sure that the food which they have just eaten passes through at once undigested. This is of course not the case. Some have one or two fluid movements after any mental excitement or emotion, and there are some whose bowels are pretty apt to want to move whenever it would be most inconvenient. In all of these cases, it is the nervous control of those functions that is at fault.

Diarrhœa may be either acute or chronic. If acute, following some sudden onset, it will usually be accompanied by vomiting and colicky pains. If the small intestine is at fault the diarrhœa of indigested food is often present.

Dysentery. Some do not understand the **difference between diarrhœa and dysentery.** Diarrhœa is a symptom. The patient, for some reason, has too frequent and too fluid movements. By dysentery is meant an actual inflammation of the bowel wall, which is evident clinically by the presence of blood, pus, and mucus in the stools. Some of the stools contain only these substances and no fæcal matter at all. In simple diarrhœa the movements are usually large and not very frequent, whereas in dysentery, especially that which affects the irritable rectum, they are very frequent, are scanty, and are passed with a great deal of pain ("tenesmus"). When the rectum is not involved there will be little or no pain and their frequency will depend on the consistency of the

stool. In amœbic dysentery (page 519), even when evidences of active inflammation are present, there may be constipation.

In the treatment of diarrhœa and dysentery the cause is to be considered. If the diarrhœa is acute, the patient is better off in bed and on a diet of boiled milk, if this is well borne, and if not, of hot gruels and soups (without condiments), milk toast, etc. Since the trouble is often due to some irritating substance in the bowel it is very desirable to give a purge, preferably castor oil, to remove this. Bismuth in very large doses is satisfactory, for it seems to have a soothing effect upon the bowel. If necessary, a little laudanum or a lead and opium pill, containing one grain of each, is given. Practically all cases of chronic diarrhœa are really cases of dysentery, and one should seek the cause. This may be an intestinal parasite, such as the hookworm (page 541), or some protozoon, as the amœba (page 520), or tuberculosis with ulceration (see below). A simple **apparently harmless diarrhœa** may be due to **as serious a condition as a cancer** in the rectum. In the case of women it is often due to trouble in the pelvic organs. These troubles of course need treatment. In very many cases, however, diarrhœa is simply a nervous phenomenon, and for that the general treatment of neurasthenia and with no local treatment, is indicated.

Ulcers of the small and large bowel develop in two diseases especially; **in typhoid fever** they cause the hæmorrhages and the peritonitis which complicate this disease (page 406); and **in tuberculosis** of the intestines, a condition of children especially, in which they give surprisingly few local symptoms. The large bowel is ulcerated in the various forms of dysentery and in tuberculosis.

Constipation, usually described as an abnormal infrequency of stools, really has as its essential feature the **abnormally long time which the food remains in the bowels,** the most conspicuous evidence of which is an abnormal **dryness** of the stools. The number of stools per week is an uncertain criterion. Most normal persons have one or two

movements a day. But, while some are very uncomfortable if for any reason they do not have one each day, others habitually go two, three or four days without a movement, their stools are not dry and they suffer no discomfort. Are they constipated?

One point it is important to remember; that a person **cannot judge of constipation simply by the number of movements** he has; the subjective symptoms also count. Some persons have regularly one large movement every day and would scoff at the idea that they were constipated, and yet they do suffer from what is called **latent constipation.** That is, the stool of each morning is not the refuse of the food eaten the day before, but of that eaten two or three days before. Again, there is an old French adage that "diarrhœa is the best symptom of constipation," and this is actually true. Some very constipated persons are quite unconscious of the fact that their colon contains masses of very hard, dry stools, which cling to its walls. These set up an irritation there which causes a diarrhœa of liquid stools past these scybalous masses. These stools often contain a good deal of mucus, which, in these cases is not a symptom of disease, as so many think, but is secreted as a protective coating to defend the bowel wall against these irritating, hard masses of stool. Such a patient will have **alternating periods of constipation and diarrhœa,** and will consider that their constipated periods are their well periods, and that the periods of diarrhœa are their sick times. If the food remains too long in the colon practically all of its water will be absorbed, and the masses may be almost as "hard as stone." These large fæcal masses usually crumble into smaller masses, giving the condition known as "sheep fæces." One or two of these lumps in a diarrhœa stool will at once give us a clew as to the diagnosis. The lower bowel may, in a severe case of constipation, become "impacted"—that is, filled by masses of hard fæces which must be removed by the fingers, or first softened by large injections of oil before they can be washed out by a water enema.

The cases of **chronic constipation** may be divided into **three groups.** First, should be mentioned the cases of **habit constipation.** Because of the careless habits of personal hygiene which lead some persons to delay each day the bowel movement as long as possible, the mucous membrane of the rectum becomes more and more accustomed to the presence of the mass of stool and after a while will fail to warn the person that it is there. This is by far the most common type. These are the patients who take so much medicine to move their bowels; who try so many food fads; who later resort to so many enemas; who use so many suppositories; all of which are unnecessary and even harmful. The more we stimulate an organ of sensation, the less sensitive does this organ become, and this is as true of the rectum as of the other organs of sensation. The more we stimulate and irritate this mucous membrane the more anæsthetic it becomes. These patients should try, by establishing the methodical habits which every normal person should have, **to restore the normal sensitiveness of the rectal wall.** If as a result of their constipation these persons have developed hæmorrhoids (piles) or fissures, these should first be corrected, since they tend to increase the tendency to constipation. The patients should try to have a movement each morning at exactly the same time; not five minutes earlier or later. Their morning routine should be somewhat as follows: on rising, drink a large cup of water; for breakfast, always a fruit, a cereal, and honey or marmalade; and then, at exactly the same time after breakfast, they should attempt, and should try hard, to have a movement. If on two successive mornings the bowels fail to move, they should on the night of the second day take before retiring a plain water enema. Above all else, these persons should use no laxative medicines. The movement of the food along their entire gastrointestinal canal is normal until it reaches the last few inches of the bowel, and the trouble there is the result not of disease but of a careless habit which should be corrected. Surely it is not reasonable to subject twenty-four feet of normal gastrointestinal canal

to a medicine which is intended to affect but one foot of it, and that the last!

In the second group of cases, **the ascending colonic group,** the seat of the constipation is the ascending colon and is due to appendicitis, gall-bladder disease especially, or other inflammatory troubles. These patients usually need surgical treatment. Certainly they should not eat a lot of coarse food, for the greater the bowel's load, the less easily can the bowel handle it. These patients may, while waiting for a convenient time for operation, take laxatives which will stimulate the bowel, as the aloes-belladonna-and-strychnia pill. Certain drugs, such as aloes and cascara, seem to stimulate the colon especially.

The third, or atonic, group includes the weak young women with enteroptosis whose entire colon, the X-ray pictures show, is too sluggish. These patients need general muscular development, such as they can gain by outdoor exercise, riding, etc., and should receive a careful diet and medication directed far more to the nervous system than to the bowel. Somewhat akin to this group are the cases of slight myxœdema (thyroid insufficiency) whose bowel torpor is an early sign of their glandular deficiency.

Other more serious causes of constipation are: liver disease, intestinal disease, acute fevers, lead poisoning, the morphia habit, dysentery, etc.

The symptoms arising from constipation are very varied. Some persons have none at all, even after a week of constipation; but most have a feeling of lassitude, headache, mental depression, or loss of appetite, and in some cases its symptoms suggest serious diseases. That these symptoms are due to any poison absorbed from the bowel, "an autointoxication" "intestinal intoxication," "colonic stasis," etc., is more than doubtful, for **such poisons** absorbed from the bowel contents **have never been demonstrated,** and the relief from a successful enema is far too sudden (within a few minutes) to be accounted for by any deintoxication; the chances are that these symptoms are mainly reflex nervous and circulatory in ori-

gin. Nevertheless, so definite are these symptoms that it is constipation that gives the patent-medicine dealers their best customers. When the label on the bottle states that the contents can cure "liver diseases, stomach disease, intestinal disorders, kidney disease, heart disease, brain disease, melancholia, in fact can make you young again," the chances are that this medicine is a simple laxative which can be taken for a long period of time; for chronic constipation can cause some of the symptoms of all these diseases and some of the symptoms of constipation have greater severity than have those due to the above diseases themselves. Biliousness means constipation much oftener than it means liver trouble, and, as for the last promise on the label, it is certainly true, for some of the most cantankerous, melancholic, pessimistic, bad-tempered hypochondriacs, become cheerful, optimistic citizens after their constipation is relieved. Diarrhoea, as has already been mentioned, also may be a symptom of constipation. The large, hard, fæcal masses in the colon often cause "neuralgia" of the sacral nerves; while one of the commonest results of constipation is hæmorrhoids, or "piles."

Acute constipation is a quite different matter, always indicating an acute and sometimes a serious disorder. Often in these cases one is tempted to give some purgative. It should, however, be remembered that **acute constipation is an early and conspicuous symptom of appendicitis and of other surgical conditions,** and that the purges so often given are very dangerous, since they may make a mild case very serious. The same is true, although in a less degree, of the acute constipation which develops early in typhoid fever. One should, therefore, not resort to a cathartic when the bowels fail to move, and especially if the patient has also such symptoms as nausea or pain, or feels "sick all over," for these symptoms may be due to the same cause as the constipation, and not be the result of the latter. Of course in the acute constipation which accompanies some acute fevers, as tonsillitis, a severe cold, etc., it is necessary to clean out the whole of the small bowel, and then calomel

or salts may be best. For the severe constipation of Bright's disease, elaterium, and even croton oil, may be necessary.

By intestinal obstruction is meant any condition which prevents the intestinal contents from flowing normally along the bowel. If it is the small bowel which is obstructed, the condition always is serious. If the colon, it seldom is serious, yet it may be, not because the bowel contents are dammed back, but because there usually is also some obstruction to the circulation of the bowel wall.

Intestinal obstruction may be due to a **tumor,** usually a cancer, growing inside and gradually filling up the bowel. **A suddenly developing obstruction** may be the first symptom of a cancer of the colon located anywhere **between the cæcum and sigmoid,** for, since there the contents of this bowel are liquid, a slowly developing obstruction will not make itself felt until its lumen is practically closed. Cancers further towards, and of, the rectum cause earlier symptoms of obstruction because there the stools are more solid. Some obstructions have been found due to foreign bodies lodged in the bowel, such as false teeth, fruit stones, etc. In still other cases the bowel is "pinched" by the contraction of the scar of a healed ulcer in its wall.

Hernia, or rupture, is one of the most common and important **causes of intestinal obstruction.** By this we mean that a loop of bowel has forced itself through one of the **weak points in the abdominal wall,** such as the navel, the femoral rings, or the inguinal rings (see Fig. 74). There are also **pockets of peritoneum** on the posterior wall of the abdomen in which the bowel may get caught, and these **internal hernias** are very hard to recognize early. The bowel is in constant peristaltic movement. The muscle fibres in rings around the bowel contract, and this constricts the lumen of the bowel (see Fig. 75); those fibres which run parallel to the lumen also contract, and this shortens the length of the loop. Waves of these two movements, passing in orderly succession down the bowel, and so coördinated that they can push its contents along, is called peristalsis.

It is an interesting fact that if a loop of bowel gets caught in a small opening, the rest of the bowel, because of these peristaltic waves, will try to follow it in. This explains why we may find several feet of bowel in one of these pockets of peritoneum, or, through an inguinal ring, outside of the

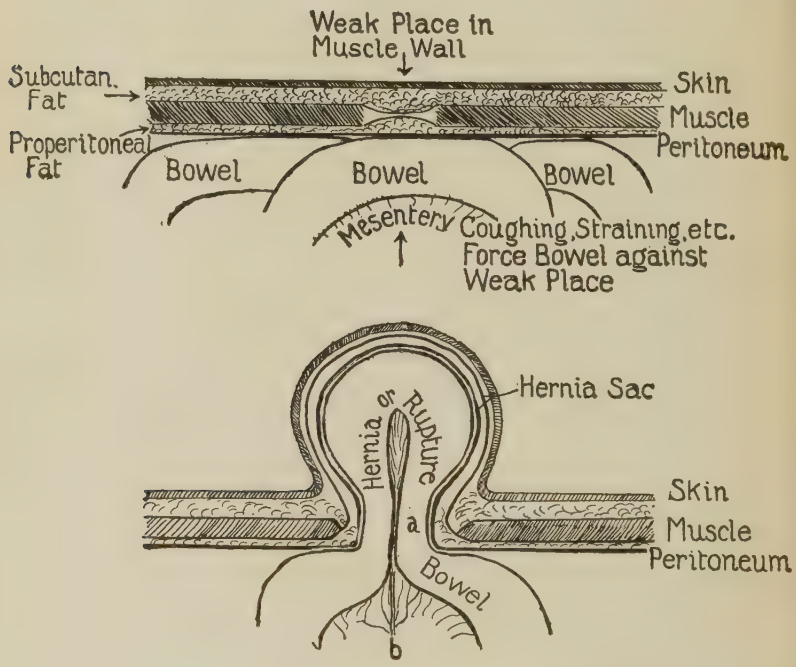


FIG. 74.—Hernia or "rupture." The weak place in the muscle wall pictured above might be of any of the "rings," or points weakened by the passage through the muscle of blood-vessels, etc. *a*, is the neck of the sac, the point where the bowel is pinched by the ring of muscle; *b*, is the artery supplying the loop of bowel with blood, and of course occluded if the pressure of the ring is great.

abdomen under the skin. If the hernia is through an inguinal or femoral ring, the patient usually can push it back easily, but sometimes it gets pinched in the opening, and he cannot push it back. Then the hernia is said to be **strangulated**—that is, so tightly pinched that its circulation is stopped (see Fig. 74). In such a case, unless relieved by operation, the bowel wall is sure to die, and the result is a fatal peritonitis.

Intussusception is a very common cause of intestinal obstruction, especially in children. By this we mean that the bowel above a certain point pushes itself into the bowel below that point (Fig. 76), much as a telescope is made shorter by pushing one section into the next, or as the finger of a

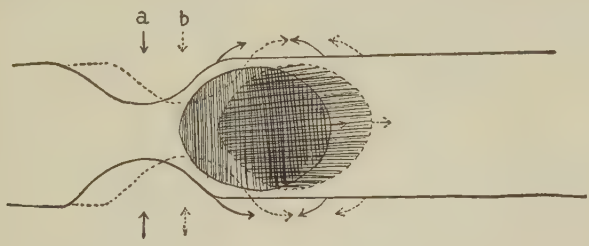


FIG. 75.—Diagram of a peristaltic wave. In the bowel is a bolus of food. The entire line, *a*, represents the beginning of the wave; the dotted line, *b*, the wave a moment later.

glove may be pushed down into its hand. Intussusception also is due to the peristaltic waves. The most common point at which this occurs is the ileo-cæcal valve (Fig. 67 and Fig. 78), and one may sometimes see the small bowel protruding

FIG. 76.—Intussusception. The bowel is represented working its way down within itself. Of course the mesentery containing the blood vessels is dragged down also and the vessels closed by pressure resulting in the death of the inner portion of the loop contained within the outer portion of this loop.

at the anus, having made its way down the whole length of the large bowel. Any child who has been seized with **pain in the stomach** and then passes **blood and mucus in his stools** should be examined to see whether or not he has such an intussusception.

And, lastly, **knots and twists of the bowel** may obstruct it. A loop may become twisted about itself, or may tie itself into a true knot. Here, also, peristaltic motion is to blame (Fig. 77).

The symptoms of intestinal obstruction are important to recognize. There is always **complete constipation.** The patient may pass blood and mucus in the stools, but **no fæcal matter and no gas.** The patient's **vomiting** is often peculiar

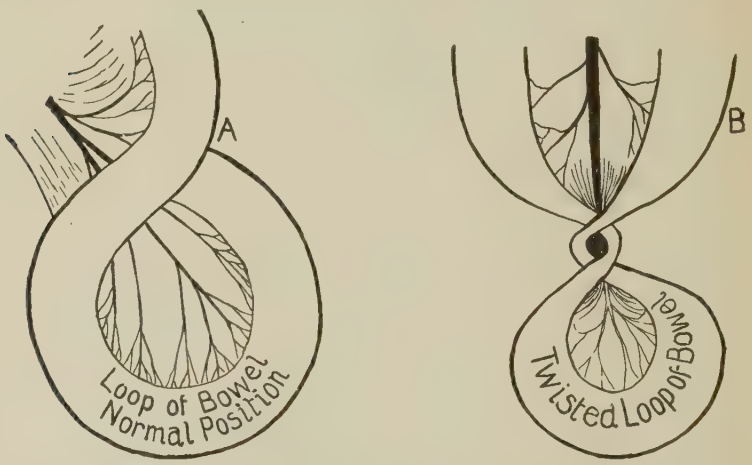


FIG. 77.—Intestinal obstruction caused by a twist of a loop. *A*, the normal position of a loop. *B*, the result of a twist. This will occlude the bowel and also close by pressure the blood vessels in the mesentery, causing the death of the bowel beyond the twist.

and unmistakable. When there is a true obstruction the peristaltic waves become very vigorous, but in the reverse direction (antiperistalsis). All the intestinal contents are now moved toward the mouth, just as normally they are moved towards the rectum. This patient vomits first the contents of the stomach, then the bile-stained contents of the upper small intestine, then the darker, worse-smelling, contents of the ileum; and, finally, if the obstruction is in the large intestine, he will vomit true fæcal masses, recognized because of their dark appearance and their foul odor. This is known as **fæcal vomiting.** The nature of the vomitus will

depend on the position of the obstruction. Only when it is in the colon can fæces be vomited. How serious the condition may become will depend on two points: first on the position of the obstruction, and second, on the circulation of the wall of the bowel at this point. In all cases the general rule is that an obstruction anywhere along the small bowel always is immediately dangerous; that an obstruction of the colon offers much less danger, and little, provided the trouble is low in the colon. But obstruction anywhere is very dangerous if this in any way interferes with the circulation of its wall. There is always pain, at first sharp and colicky, and later continuous. If the obstruction is of the small bowel, soon the patient may show collapse, and then follows death. If the obstruction completely stops the circulation of the loop, that loop of bowel dies and becomes gangrenous and a fatal peritonitis sets in. In obstruction of the lower portion of colon, the symptoms are much slower in their development. The constipation may last for days, the abdomen become very much swollen, loops of bowel show themselves under the abdominal wall, and fæcal vomiting occur; but since in these cases the circulation in the bowel wall iss eldom interfered with the symptoms of peritonitis are absent, or appear very late.

The treatment of intestinal obstruction is usually surgical. The doctor never gives a purge, for this will only make matters worse. If the trouble is an external hernia, the bowels may perhaps be pushed back. If no surgeon is at hand the patient is kept flat on his back, with the knees flexed, and an ice-bag kept continuously over the lump. This position and the effect of the cold may relax the opening and allow the bowel to drop back. If this does not happen promptly, the trouble should be treated surgically. Sometimes, even when serious, the trouble does right itself without operation, but so rarely does this occur that the risk should never be taken.

By enteroptosis is meant, literally, that the intestines have fallen, but the word usually means that the stomach (gas-

troptosis, see page 190) intestines, liver, kidneys, and all the viscera (visceroptosis, see page 190) have, for some reason or other, sagged to a position too low in the abdomen. One sees this in two groups of patients. In the first group are **women who are the mothers** of several children, and also those patients whose abdomen, because of heart or liver trouble, has previously been greatly distended with fluid. The enteroptosis in these cases may be extreme, but, strange to say, these patients as a rule complain of no symptoms whatever. But enteroptosis is met with also (the second group) **in young nervous persons,** especially women, and they complain of a variety of severe symptoms, such as dyspepsia, dragging pain, throbbing of the aorta, etc., although in these cases the actual displacement of organs is less than in the first group. The treatment of the second group is to insist on normal life in the open air, with whatever outdoor sports are possible, and on a full diet, so that the patient will gain weight (a little adipose tissue in the peritoneal cavity is by all means the ideal abdominal support). One must of course treat the neurosis, and may support the organs by proper binders as long as this gives relief.

Mucous colitis is almost always a nervous manifestation, although very rarely it is due to some local trouble in the pelvis. Patients with this trouble complain that they pass slime with their stools, and that sometimes the whole stool is nothing but mucus, although as a rule it is mixed with fæcal matter. Sometimes the mucus is passed in long strings, sometimes in long tubes, just as it had peeled itself off the wall of the bowel. These patients are sure that they have a tapeworm, or that a portion of the bowel itself has been passed. They suffer, usually, severe colicky pains produced while the long sheets of mucus are peeling off. These patients usually have also at times nervous diarrhœa and many other neurotic symptoms. Their **suffering is often extreme,** and they are frequently operated on for appendicitis, gallstones, pelvic trouble, or intestinal adhesions. We not infrequently see a patient who has had all these operations per-

formed. It is a general rule in medicine never to advise an abdominal operation on a woman who lately has passed mucus in her stools, no matter how severe her abdominal pains, unless there are also other clear indications for operation. Since, as a rule, these patients are very constipated,

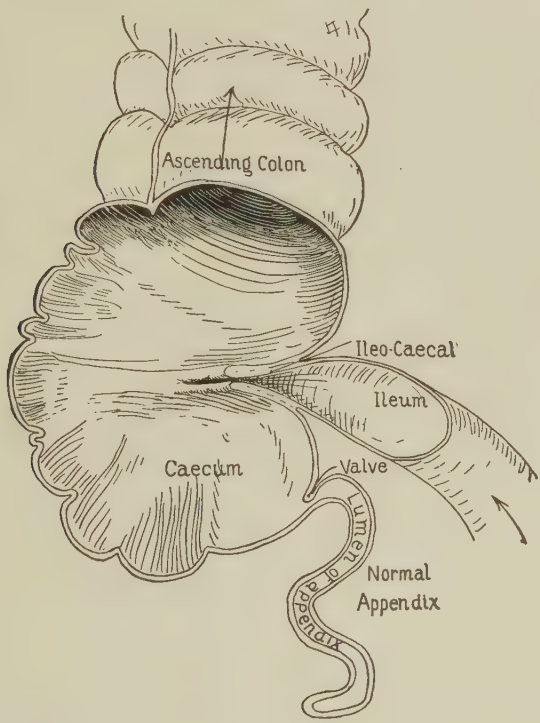


FIG. 78.—The normal cæcum showing the ileo-cæcal valve and the appendix vermiformis.

this should be treated, by diet especially, exercise, physical therapies, etc. But the chief treatment necessary is that for the neurosis (page 556), which means education concerning the condition, encouragement, full diet, and, later, occupation.

By infarction of the bowel, as described on page 89, we mean that one of the blood vessels supplying the bowel has for some reason become plugged, and hence the bowel wall,

in the area supplied by this end artery, will soon die. Unless operation is performed and the infarcted portion of the bowel removed, fatal peritonitis may result.

The symptoms of intestinal infarction (mediastinal thrombosis) are similar to those of obstruction, except that the

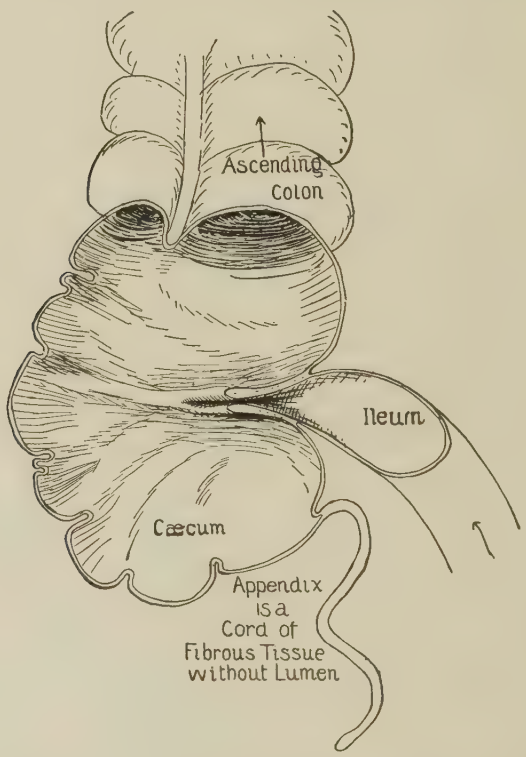


FIG. 79.—Chronic obliterative appendicitis. As a result of repeated slight inflammations the appendix is a solid cord of scar tissue.

patient with the former passes more blood in the stools. It occurs most often in heart disease and in any other condition which favors embolism or thrombosis. This condition is much more frequent in horses ("colic") than it is in men, but in the case of the former it is due to parasites which live in the blood vessels of the intestine.

The Appendix

The appendix (Fig. 78) is a small piece of bowel about four inches long and as large around as the little finger. One end of it is closed, the other opens into the cæcum just below the ileo-cæcal valve. It lies free in the abdominal cavity. No definite function for it can be assigned. It certain fills with food and empties as regularly as the cæcum.

Chronic inflammation of the appendix is not at all uncommon. As the result of repeated, mild, acute attacks, its wall gets thicker and thicker, and its lumen narrower and narrower, until finally the whole appendix is nothing but a thick, solid cord of scar tissue. This condition is known as **chronic obliterative appendicitis** (Fig. 79). Strange to say, the chief symptoms of this condition are not in the appendix or anywhere near it. Often they are gastric, and the patients complain of "hyperacidity," of heart burn, of dyspepsia; while many have repeated attacks of vomiting. All of these symptoms are probably due to reflexes from the appendix. Some of these patients have also severe constipation which will not yield to ordinary treatment, and sometimes attacks of severe abdominal pain, some of which probably is due to the efforts of the thickened walls to expel the masses of fæcal matter from its narrow lumen. Nearly seventy per cent of the **cases of chronic appendicitis** have also **chronic inflammation of the gall-bladder,** cholecystitis, which often is more important than the appendicitis. Various reasons are given for this association; that the conditions favoring the infection of the one favor that of the other also; and that just as the reflexes from the appendix disturb the normal functioning of the pylorus so they disturb the functioning of the little valve in the duodenum which opens the gall-bladder, causing stagnation of the bile in this receptacle. In the case of the appendix it is this stagnation of its contents which aids us so much in the Roentgenological diagnosis of an abnormal appendix. If, twenty-four hours after the barium meal, the appendix still contains barium, while the cæcum has emptied itself, but also, if at that time both cæcum and

appendix contain barium and the cæcum is not freely movable when palpated, then very certainly there is disease in and around the appendix.

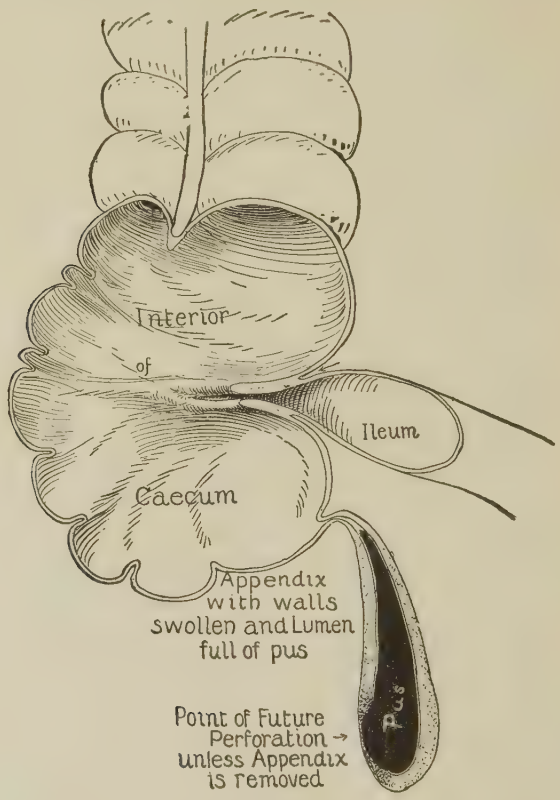


FIG. 80.—Acute appendicitis. If operation is performed now the wound can be "closed" and in about a week the patient will be up and about. But if the operation is not performed, the appendix in a few hours will rupture and local (leading to abscess) or general peritonitis will follow.

Acute appendicitis (Fig. 80) may give symptoms so slight that final judgment is almost impossible. One may say that it often begins with a **general stomach-ache, followed by vomiting**, and this by **tenderness on pressure over the lower half of the right side of the abdomen.** Just how much tenderness there will be, whether or not there is constipation or

diarrhœa, pain on defecation or on micturition, vomiting, or hiccough, will depend not so much on the appendix itself as on just where it lies, and it certainly is curious in what a variety of locations its tip may be found.

It is surprising how many cases of mild, acute, or subacute appendicitis will for years **without local symptoms,** masquerade under the picture of some other disease, as dyspepsia, or even as chronic constipation. The history of these symptoms, or of frequently recurring abdominal pain, should always lead the patient to seek careful examination.

Outspoken acute appendicitis is most apt to develop in an appendix already the seat of previous, very mild attacks. Acute appendicitis is always caused by some germ which settles in the wall of the appendix, but in a definitely acute case this germ is virulent enough to set up there an inflammation which leads either to gangrene of the wall or to pus formation. If the appendix can be removed before the inflammation has gone through its wall, then there is no further trouble; but if we use medicinal treatment in the hope that it will subside (as it does in many cases), there is then a good chance that the inflammation will affect the surrounding loops of bowel causing general peritonitis, or, if less virulent, an appendix abscess (Fig. 81). These acute cases will from the first have fever and a leucocytosis. If the operation is performed before local peritonitis has set in, the abdomen can be closed at once, and the patient be out of the hospital in ten days; but in the latter case a drain of rubber tube must be put into the wound to allow the escape of the exudate, and the patient will remain in bed, sometimes for six weeks. One never knows just what course such an infection will take.

If the infection is very virulent, the wall of the appendix **may become gangrenous and perforate,** or the appendix fill with pus and then rupture, setting up a general peritonitis within less even than twelve hours after the first pain. If the process is not so very rapid, the loops of bowel around the inflamed appendix will, because of a slow peritonitis, become

stuck together—"adherent"—and form a wall against the spread of the trouble. Within this enclosure a large abscess may form which contains the pus and perhaps also some intestinal contents. The trouble is now a local peritonitis,

FIG. 81.—Acute appendicitis, gangrene of the appendix, perforation, and spreading peritonitis. In this case the appendix was not removed, and the inflammation has spread to the surrounding loops of bowel. An abscess will form in the shaded area or the inflammation may spread through the entire peritoneal cavity.

that is, an **appendix abscess.** These abscesses may contain even a quart of bad-smelling pus. Since we never can know until we operate just what is happening in an inflamed appendix, it is always better to operate at once, since the earlier the operation the less the danger. While the ques-

tion of operation is still undecided, morphia is never given, no matter if the patient does suffer, because we need the patient's help by describing his symptoms and this means a perfectly clear mind. True, many cases do get well without operation, and sometimes there is no surgeon near. If that is the plan, the patient should remain absolutely quiet in bed and receive no nourishment by the mouth. Under no circumstances should he take a purge, for this would make the trouble all the worse, and greatly hurry the disease. He should from the first be made quiet and comfortable with morphia, with ice bags lying against the abdomen.

During the last few years **epidemics of appendicitis** have attracted considerable attention. The cases in these epidemics are often very severe and the appendices removed found highly inflamed and even gangrenous, thus ruling out completely the suspicion that fear may have led to many unnecessary operations. The explanations given for such epidemics is that the germ which causes the appendicitis and which must of course reach the blood stream through an external portal of entry which itself is subject to epidemic inflammation, as the tonsils, is one with a special affinity for the appendix tissue, for there is but little doubt that a germ which one patient gets from another, whatever the route, tends to attack the same tissue of each patient it reaches.

That subacute appendicitis, like subacute rheumatism, subacute nephritis, or endocarditis, resembles a smouldering fire which frequently flares up because of reinfection from some latent focus of suppuration, is now well understood, and therefore it is quite as important to operate on this portal of entry (tonsils, nose, teeth, etc.,) as it is to operate on the appendix itself. In women the appendix also is usually inflamed when there is an inflammatory disease in the pelvis, and, vice versa, when appendicitis is the primary trouble.

People very often ask why we have so much appendicitis now, while a few years ago it was unheard of, and the answer

is that formerly we heard of many who died of "inflammation of the bowels," of "general peritonitis," and of "abscess of the intestines"—conditions which result from neglected appendicitis. Now, when a case just begins, since we have no means of telling whether it will subside without further trouble, or will lead to chronic trouble, or whether within twelve hours there may be a general fatal peritonitis, we have learned to choose early operation as always the safer course. Nevertheless, statistics show that **appendicitis each decade is a more common disease** than it was during the former, partly because a large percentage of the population now reach adult age, but more because of our too purified diets and the very sedentary habits of modern life.

Meckel's diverticulum, a tube which sometimes persists on the lower ileum and which much resembles the appendix, is a relic of fœtal life. This, if inflamed, may cause attacks similar to appendicitis and require similar treatment. The pain and tenderness in these cases, however, usually are to the left of the umbilicus.

Peritonitis, or inflammation of the general abdominal cavity, is one of the most dreaded of abdominal conditions. As a rule it begins as a strictly local condition and its severity follows the general rule that the nearer the diaphragm it starts the more dangerous it is. Without mentioning in detail those forms of peritonitis due to bullet wounds and stab wounds of the abdomen, it may, in the upper abdomen start from a perforated gastric or duodenal ulcer, or a rupture of the gall-bladder; these cases are very serious. Lower down, it starts especially from a ruptured appendix or a perforated intestinal ulcer (typhoid, tuberculosis, or amœbic); while peritonitis from a low-lying appendix or from an infected Fallopian tube is the most common form of all; but with the exception of those cases, always serious, due to the pneumococcus, the organism which also causes pneumonia, these pelvic cases remain local because nature quickly walls them off. **The symptoms of general peritonitis are:** general abdominal pain, fever, and vomiting. The patient lies on his back with his

knees flexed (to loosen the abdominal wall and to hold the bedclothes off the abdomen). The abdomen is sooner or later swollen, does not move at all on respiration, and is very tense and tender when palpated. The bowels do not move and no gas is passed. The blood usually shows a high leucocytosis. Later, the face becomes pinched and pale, the patient sinks into collapse, becomes unconscious, and dies.

The treatment, if early, is to operate at the point of origin of the peritonitis, allowing drainage at least at that point. The patient is propped up in bed to allow the exudate to settle towards the pelvis, is kept comfortable with morphia, and is given normal salt solution and glucose by vein or by rectum. Sometimes he gets well. Tuberculous peritonitis, a very distinct form, is described on page 455.

CHAPTER XI

Diseases of the Liver, Gall-Ducts and Gall-Bladder

Anatomy

The liver is a large organ, weighing about 1500 Gm., or three pounds. It is, for the most part, tucked away behind the ribs in the upper right-hand part of the abdomen.

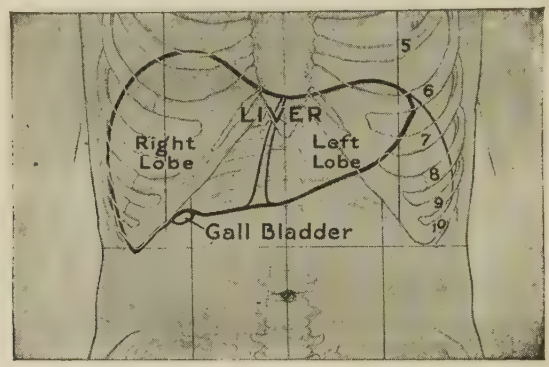


Fig. 82.—Outline of the liver, anterior view.

The liver is made up of a myriad of small livers called **lobules** (Fig. 83), each just about large enough to be seen with the naked eye, practically all of them similar in size, shape, and function. Study one of these little livers, and we study the whole organ. Each lobule has somewhat the shape of a thimble (Fig. 84). It is composed of liver cells and vessels. Through its centre runs a tiny tributary to the hepatic vein, *a*, which carries the blood to the heart. Along the outside of each lobule run three or four tiny branches of the portal vein, *b*, which brings the blood from the digestive organs to the liver. These portal

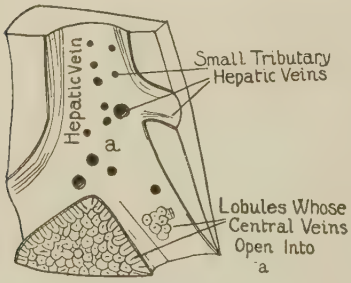


Fig. 83.—A small fragment of liver very slightly magnified. The cut was made along a small hepatic vein. In the lower portion the lobules are represented.

and hepatic veins are connected by a multitude of small capillaries, *c*, and around these the liver cells are arranged. The liver is, therefore (Fig. 85, *d*), composed of multitudes of cells on one or several sides of each of which is a capillary, *c*. Through these capillaries flows practically all the blood from the stomach, bowels, pancreas, and spleen, so that every particle of food which is absorbed by the blood has to pass

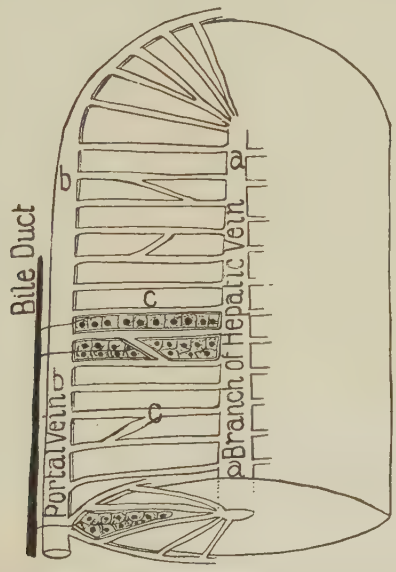


FIG. 84.—One liver lobule, in vertical section. The blood flows from *b*, a branch of the portal vein, through the capillaries, *c*, to *a*, a branch of the hepatic vein. While in the capillaries it comes in contact with the liver cells. From these cells start the bile capillaries which flow to the bile duct.

by these liver cells. From these same cells start also tiny bile ducts, the tributaries of the gall-duct (Fig. 85). In addition to these characteristic liver cells are others arranged along the capillaries, the reticuloendothelial cells, which are very phagocytic, and which would seem to aid in removing and destroying the worn out red cells ready to be retired from circulation, conserving their hæmoglobin, splitting it, saving the iron-containing fraction for new hæmoglobin, and aiding in the manufacture of bile pigment from the other fraction.

Physiology

The functions of the liver are various. Its **glycogen-storing activity** is very important. We often eat several ounces of carbohydrates at a meal, most of which is transformed in the stomach, the small bowel, and bowel wall, into glucose, and then collected in the blood of the portal veins. Were it not for the liver, and the muscles which are almost as important as the liver, this glucose would at once enter into the blood of the general circulation, which, as a result, would at times be far too rich in sugar, and at other times too poor. The liver removes from the portal blood practically all of the glucose which the blood has collected from the intestine, changes it into animal starch, or "glycogen," stores this up, and then, as occasion, requires, doles it out again to the blood as glucose. The liver often contains about one third of a pound of glycogen—rather a large amount, considering that the circulating blood of the entire body does not contain at one time over one third of one ounce. Some of the glucose also is transformed into fat and stored up in other depots of the body. Glucose is the coal of the body, of which the circulating blood must always contain a small and constant amount, but not much over 0.1 per cent. (100 mg. per 100 c.c. of blood). When the ability of the body to use glucose is lowered, and, as a result of this, the general circulation contains a surplus, the kidneys remove this from the blood and excrete it in the urine.

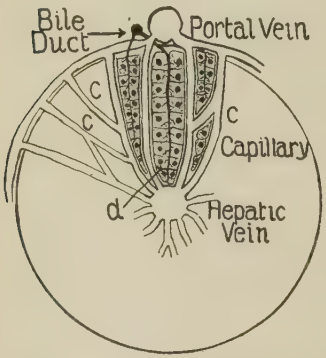


Fig. 85.—One liver lobule in cross section. The blood flows from the branch of the portal vein through the capillaries, *c*, to the branch of the hepatic vein. While in the capillaries it comes in contact with the liver cells, *d*. In these cells arise the tiny bile capillaries which empty into the bile duct.

Again, all the food of the body and all of the living protoplasm itself are sooner or later burned up, and their ashes excreted through the kidneys. Just what all these ashes are is not known, but some, especially ammonia bodies, we

know to be poisonous. If they were allowed to remain in the blood until the kidneys could get rid of them all, the body would suffer. It is one of the liver's functions temporarily to **remove these ashes from the blood and to transform them** into something—chiefly into urea—which is not poisonous, and then to return this to the blood. This the kidneys then eliminate from the body.

A third function of the liver is to **remove from the blood certain ashes and to excrete them in the bile.** The bile pigment, made up of used-up hæmoglobin, is one illustration. Cholesterol and many other waste products also are eliminated in the bile.

Further, the bile is, in a certain sense, a digestive fluid. **The bile acids,** which give vomitus its bitter taste, probably are not excreta, but are manufactured by the liver to aid in the absorption of fats and soaps, for these, though not very soluble in water, are soluble in the bile. These bile acids must greatly aid the mucosa, for, when a gall-stone prevents bile from entering the intestine, only about one fifth of the fat of the food is used, and the rest is lost in the stools. The bile acids also keep the cholesterol in solution, and this is important, because cholesterol, when it is no longer in solution, forms gall-stones. Much of these bile acids is promptly reabsorbed by the bowel and is again eliminated by the liver, perhaps several times, thus travelling in a circle. One reason for the importance of this is that these bile acids are efficient stimulants to hepatic functions. That is one reason why we prescribe them as components of cathartic pills.

The liver **has two secretions, an internal secretion** of glucose, urea, and perhaps many other substances, which it turns back into the blood; and an **external secretion, the bile,** which contains two groups of bodies; the secreta, or substances manufactured for a specific purpose, such as the bile acids; and the excrementa, or substances no longer of any use to the body, which are merely got rid of through this route. About 500 to 800 c.c. of bile are secreted every twenty-four hours.

From popular talk one might infer that liver trouble was rather common. One hears so often the word "bilious," and of remedies to "stir up the liver"; but the symptoms of which "bilious" persons complain are almost invariably due to the gall-bladder or the colon. There are many diseases of the liver with severe symptoms, but, strange to say, these symptoms do not always suggest the liver as their source.

Diseases of the Gall-Ducts and Gall-Bladder

Jaundice is a common symptom of liver trouble, but more often of troubles in the gall-ducts. The little bile ducts start in the liver cells (Fig. 85), then unite, forming larger ducts, which continue to unite until we have the large **hepatic duct,** a tube about two inches in length and a quarter of an inch in diameter. Underneath the liver is the **gall-bladder** (Fig. 68), a pouch holding about 30 to 50 c.c. of bile, with a duct, the **cystic duct,** which is about half an inch long, and an eighth of an inch in diameter. The hepatic duct and the cystic duct unite together to form the **common duct,** a tube about three inches long and about a quarter of an inch in diameter, which opens into the intestine. The liver is practically always forming bile, but between meals much of this bile is stored up in the gall-bladder. It is not stored up, however, as secreted, but is concentrated by the wall of the gall-bladder by removal of its water, until it is from five to ten times more concentrated than when secreted. After a period of fasting, as soon as fatty food begins to pass from the stomach through the pylorus and there is immediate need of bile, the bladder empties this reserve through the common duct into the duodenum.

If the bile cannot flow into the intestine, but is dammed back into the liver, it is reabsorbed into the blood, is carried over the whole body, and tinges the skin, the whites of the eyes, and the urine, to a yellow or a greenish yellow. This condition is called "jaundice." It has been customary to speak of two forms of jaundice, the hepatogenous and the hæmatogenous. **Hepatogenous jaundice** is due to obstruc-

tion in the hepatic or common gall-ducts. This may be caused by a plug of mucus in the ampulla of Vater (the opening of the gall-duct into the duodenum), by inflammatory swelling of the mucosa of the duodenum, or by a stone lodged in the common or hepatic ducts, or by a tumor pressing against these ducts and closing them. A gall-stone plugging the cystic duct will of course not cause jaundice, because that will not dam the bile back into the liver.

In hæmatogenous jaundice the trouble is also an obstruction to bile flow, but in this case the obstruction is in the finest gall-ducts. Perhaps there is so much solid matter excreted in the bile that this becomes too viscid to flow, clogs the smaller ducts, and so dams the latter, forcing bile back into the liver cells and hence into the blood. Hæmatogenous jaundice is seen particularly after a great destruction of red blood cells, such as occurs in severe fevers, malaria especially, but also typhoid fever, pneumonia, lues, etc.; in any severe toxic condition which causes destruction of liver cells, such as acute yellow atrophy; or as the result of certain poisons, as chloroform, phosphorus, and arsenic, especially arsphenamine. Additional illustrations of hæmatogenous jaundice are the hereditary jaundice, which may affect several children of the same family, and the jaundice of the newborn, which may be fatal, but which usually disappears in less than two weeks.

The most conspicuous symptom of jaundice is the color, which, when very slight, is best recognized in the whites of the eyes. If there is total obstruction of the bile flow, the skin may be almost any shade of yellow, green, or even brown. The stools will be white or **clay-colored,** for they normally owe their color chiefly to the bile pigment. The bile in the blood stream is poisonous and affects the heart, so that the **pulse is slow,** and the **blood coagulates tardily.** This is why surgeons hesitate to operate on jaundiced patients until they have increased the coagulability of the patient's blood.

Catarrhal jaundice is a simple jaundice which occurs com-

monly in young persons and which lasts from four to eight weeks. There are, as a rule, very few symptoms, other than the jaundiced skin and the clay-colored stools. A few cases, however, have a slight fever, nausea, loss of appetite, intense itching of the skin, slow pulse, and mental torpor. In the majority of cases the original trouble is **a slight inflammation of the mucous membrane of the duodenum,** which not only closes the mouth of the common duct, but which extends up along the bile ducts into the liver, sooner or later affecting the whole liver. Some cases, on the other hand, would seem to start in the liver. This condition has developed as an epidemic disease.

The treatment is simple. The patient should be kept quiet, the gastroenteritis treated by a light diet which, because of the lack of bile in the bowel, should contain very little fat. The patient should drink much alkaline water, and the bowels should be kept open by mild salines. The use in catarrhal jaundice of the Lyons method of draining the gall-bladder has proved of value.

Simple obstructive jaundice without pain may, in elderly persons, be a serious matter, since so often it is due to a tumor in the pancreas or the duodenum. Courvoisier's law, often quoted in this connection, is as follows: if an adult develops an obstructive jaundice and the gall-bladder is enlarged, one may suspect that the common duct is closed by a cancer in the head of the pancreas (through which this duct flows), or in the ampulla of Vater; if, however, the gall-bladder is not enlarged, one may hope for some benign cause of obstruction, as a gall-stone.

By acute cholecystitis is meant an inflammation of the gall-bladder wall, due to bacteria. In about one half of all cases these bacteria are either the colon bacillus, or the typhoid bacillus. Infection of this pouch is very common in a great variety of infections, e.g., in sore throat, and in many acute fevers, particularly typhoid. It is particularly common if gall-stones already are present, and results from any condition which causes sluggishness of the bile flow

from the bladder, such as rest in bed, pregnancy, abdominal tumors, etc. The frequent association of an infected appendix and an infected gall-bladder is mentioned on page 221. This explains why so many persons whose appendix was removed a few years before, but without relief, must now have the gall-bladder removed or drained. One explanation made is that the appendix trouble inhibits the emptying of the gall-bladder, thus causing stagnation of bile in this organ.

In mild cases of cholecystitis there may be but a few slight symptoms suggestive of this condition: chilly feelings, fulness and weight in upper abdomen, heartburn, nausea, gas and acid eructations, and many others usually counted as dyspepsia, all of which usually quiet down as a result of rest in bed, liquid diet, and the local application of an ice bag. More severe cases, with symptoms including fever, severe pain, tenderness over the right rectus muscles, vomiting, and an easily palpated bladder, require greater watchfulness; but if the gall-bladder becomes full of pus the condition demands immediate surgery.

The chances are that it would be much better for those patients with mild cases of gall-bladder trouble were surgery more often resorted to, since an infected, even though quiet, gall-bladder can do an immense amount of harm. For the gall-bladder is a **very suitable home for germs.** After an attack of typhoid fever, the typhoid bacilli can live there for perhaps forty years, and streptococcus viridans for months or even years, and persons harboring these bacilli, themselves feeling perfectly well, spread the disease broadcast.

Other **chronically infected gall-bladders,** even though they contain no gall-stones, **may cause very pronounced symptoms,** but not those which suggest the real trouble. In fact, the causes of those symptoms generally called "dyspepsia"—that is, heavy feelings or pain after eating, sometimes very severe heartburn, the eructation of gas or of acid fluid, nausea and vomiting, are, most of them, the results of reflex pylorospasm. Subacute cholecystitis ranks first, explaining twelve times as many cases as do gastric ulcers, twice as

many as do duodenal ulcers, and three times as many as does subacute appendicitis. Chronic cholecystitis is suggested as a cause of dyspepsia in any case of dyspepsia which develops in middle life, especially if the patient has had typhoid fever years before and with slow convalescence, if the patient is obese, and, if a woman, if she is a multipara.

Gall-stones are made up chiefly of cholesterol (page 231) and bile pigment (page 229), sometimes of pure cholesterol, sometimes of both mixed, but the most of them are impregnated with calcium salts. They vary in size from that of a grain of sand to that of a pigeon's egg or even a hen's egg. Some are so soft that they can be crushed in the fingers, others are hard as rock. They form chiefly in the gall-bladder, but may also form in the larger gall-ducts when these are dilated. The gall-bladder may contain only one, but as a rule it contains a dozen or even two or three hundred stones. Gall-stones occur especially in the bladder of those who have had typhoid fever, in those who gain weight rapidly in middle life, and in women who have borne several children. Germs and the stagnation of bile would seem the predisposing factors, but in some cases the simple precipitation of cholesterol would seem the cause. The great wonder is that gall-stones are not commoner, for cholesterol, of which there is much in the bile, is extremely difficult to keep in solution, and especially when it is increased in amount, as it is said to be by infections of the liver, as during the convalescence of typhoid fever, and by pregnancy. Given a nucleus of bacteria or a little flake of mucus, and around this will crystallize readily the cholesterol and bile pigment.

The symptoms of gall-stones vary greatly (Fig. 86). In the first place, a person may have the gall-bladder full of them and never know it, provided the stones stay quiet, although their presence would encourage attacks of subacute cholecystitis (page 234), but these will, as a rule, be interpreted as "dyspepsia" due to other causes. They always cause a slow, chronic thickening of the gall-bladder wall, which contracts down on the stones, holding them tight, and

ending all usefulness of the gall-bladder as such. But if one of these stones becomes carried by the current of bile into the cystic duct, then the patient knows something is the matter. If the stone is tiny, the patient may have an attack of "acute indigestion" with nausea and vomiting, which

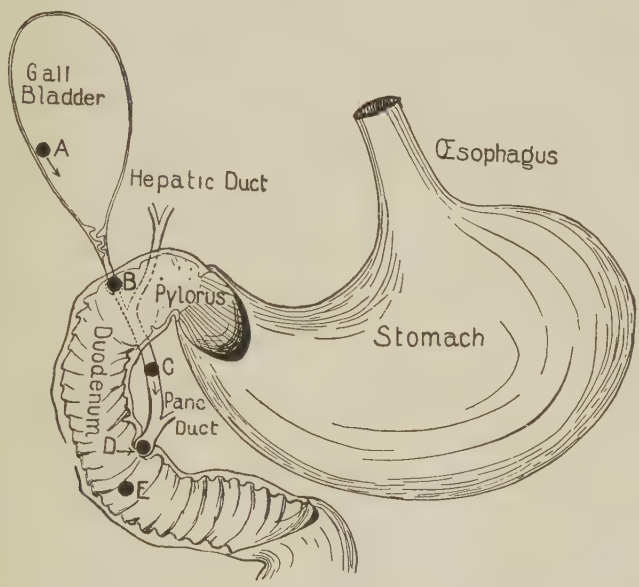


FIG. 86.—Outline of the stomach, duodenum and bile ducts showing the various possible positions of gall-stones. *A*, gall-stone in the gall-bladder, which will cause no symptoms. *B*, stone in the cystic duct which will cause colic but no jaundice. *C*, stone in the common duct. This will cause less pain than *B*, since the duct is larger, but will cause jaundice. *D*, stone in the ampulla of Vater. This will cause "hepatic intermittent fever" and jaundice, and also will disturb the pancreatic functions. *E*, the stone has reached the bowel. It will cause no symptoms.

though very explosive in character often causes little or no pain. But if the stone is large enough to have some difficulty in forcing its way down the cystic duct, the patient has "gall-stone colic." All of a **sudden, like "lightning from a blue sky," he feels an extremely sharp, severe pain,** running from the right side under the ribs to the right shoulder-blade. The patient sometimes rolls around on the floor, vomiting, and screaming with excruciating pain. This pain

is caused by the passage of the stone, not only as it squeezes its way through the cystic duct, but also as it passes through the common duct, although this duct is about three times as big as the cystic duct. At the mouth of the common duct the stone reaches the ampulla of Vater (Figs. 86 and 93). This is a pouch in the mucous membrane of the duodenum into which open the common bile and the pancreatic ducts. The opening into the bowel is through a tiny hole. The stone now either forces its way through this hole into the duodenum, in which case the pain of course ceases at once, or it stays in the ampulla for months, producing the symptoms of a **stone in the common duct.** The ampulla is roomy and allows the bile to flow into the bowel fairly freely around the stone; but occasionally, perhaps due to a slight swelling of the bruised mucous membrane, the stone entirely plugs the orifice. The patient, who has been slightly jaundiced ever since the stone reached the ampulla, then has acute attacks of sudden, severe pains in the upper abdomen, during which the jaundice gets much deeper, the stools clay-colored, and the fever rises rapidly with a chill and sweat. After a few hours, the pain rapidly subsides, bile again flows into the intestine, and the jaundice somewhat clears up. This condition is often mistaken for malaria. It is known as the "intermittent hepatic fever of Charcot."

Some large stones are not able to force their way into the gall-ducts. Some try, cause a dull ache "in the pit of the stomach," which, however, does not radiate, and then fall back from the neck of the gall-bladder into its roomy cavity, and the pain ceases. These patients are never jaundiced. Other stones set up in the gall-bladder an inflammation which extends through to a loop of bowel, or to the abdominal wall; then ulceration follows and a fistula forms through which the stone escapes into the bowel or through the skin.

The chronically infected gall-bladder, however, whether it contains stones or not, really causes a great deal more trouble than pain, since it is **the most important cause of the chronic indigestion** described on page 181, and in addition

may provide some of the germs which cause more general infections (see page 235). It also causes reflex disturbances of the action of the heart and lungs. Though it may not be the original cause of these intrathoracic troubles, yet many cardiac irregularities and some asthmatic attacks are relieved by operation on the gall-bladder.

The diseases just mentioned are really diseases of the gall-bladder and gall-ducts, not of the liver, although, of course, the liver always does suffer. The diagnosis of gall-stones is easy when the attacks are a typical colic, but in other cases it may be very difficult, which is natural, since these digestive disturbances mentioned are largely the result of pylorospasm, and this may be caused also by gastric and duodenal ulcer, and by subacute appendicitis. The X-ray plates are convincing for diagnosis when the shadows of the stones are clearly seen, but this means they contain considerable calcium and by no means is this true of all gall-stones, especially those of recent origin. As a matter of fact, but few of the gall-stones are visible on the films. More are seen as negative shadows on the plates taken of gall-bladders made visible by the Graham method.

The treatment of ordinary gall-stone colic is to make the patient as comfortable as possible by putting him into a hot bath and administering either chloroform or morphia. A person subject to gall-stone or gall-bladder attacks should be on a diet as methodical as possible; that is, the stomach should never be "surprised" by meals which are at all unusual in time, quality, quantity, flavor or temperature. Especially, he should avoid an excess of starches, sugars and fats. The surgery of the gall-bladder is now so perfect that one may safely recommend its operative drainage or removal. The surgeon will decide which after he sees the condition of its wall. Such an operation will, in about ninety per cent of the cases, prevent any recurrence of the pain, and will conserve the health of other organs. If the stone is lodged in the common duct, however, an operation is imperative, even though dangerous.

Transduodenal drainage of the gall-bladder through a Reyfuss stomach tube (Lyons' method) certainly does give relief. The tube is allowed to remain in the stomach until it passes into the duodenum, as shown by the fluoroscope or by the alkaline reaction of the aspirated fluid, then fifty cubic centimeters of saturated magnesium sulphate solution are introduced. This would seem to relax the sphincter at the mouth of the common bile duct, and allow the escape of its contents. First, the bile of the common duct escapes; then, and with a

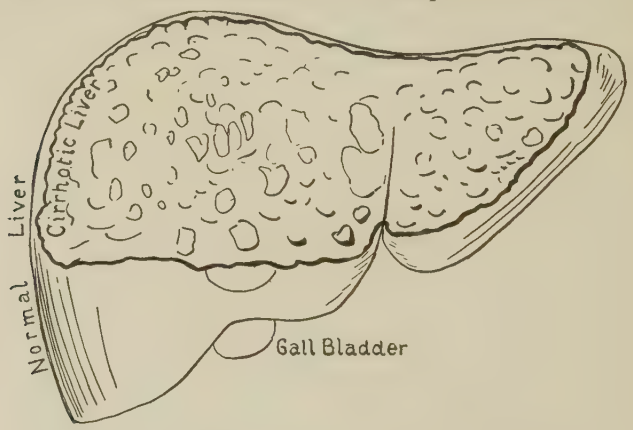


FIG. 87.—The outline of a normal liver and of a cirrhotic or "hob-nail" liver to show their relative sizes, and differently appearing surfaces. (Much reduced.)

transition which is sudden, that of the gall-bladder, then, that from the hepatic duct. Patients thus treated each seven or ten days seem to get considerable relief.

DISEASES OF THE LIVER

Atrophic cirrhosis of the liver is a disease important to have in mind when dealing with doubtful conditions of middle life. It is due to various poisons—particularly alcohol, that injure the liver cells, which gradually diminish in size, and die. Their place is taken by scar tissue, and scar tissue always contracts. In time we may have in the liver more scar tissue than liver cells. The result is at first a large liver, then a small one, which may weigh only from one to one and a half pounds. It is very hard, for it now consists chiefly

of fibrous tissue. Also, its surface is very rough, because the scar tissue within it is in rather coarse bundles, which, by contracting, pull in the capsule in certain points and cause the islands of normal liver tissue to bulge out in little lumps, hence the name "hobnail liver" (Fig. 87).

Symptoms of cirrhosis of the liver, due to a diminishing amount of liver tissue, do not appear until late in the disease,

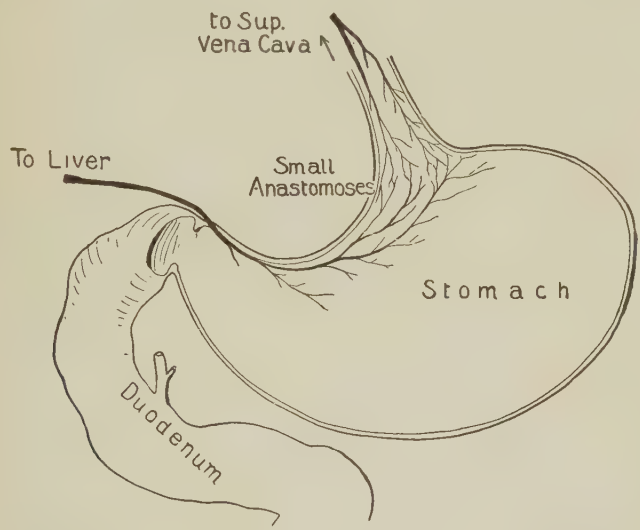


FIG. 88.—Diagram of the stomach showing the venous circulation at the lesser curvature. It will be seen that the blood in the veins of the œsophagus flows upwards and empties into the superior vena cava without flowing through the liver, and that the blood in the veins of the lesser curvature of the stomach flows towards the pylorus and then through the liver. These two sets of veins are united by fine anastomoses.

for a small amount of liver is enough to perform all the functions of this organ, although slight digestive disturbances are common; loss of appetite, flatulence, nausea, etc. The first symptoms are due to disturbances of the portal circulation. Practically all the blood from the digestive organs is collected in the portal veins and carried to the liver. Since these cirrhotic livers do not allow it free passage, it is dammed back into the digestive organs, with the result that they become the seat of **chronic passive congestion** (see page 120);

that is, they are choked up with the blood and hence cannot functionate well. These patients have "gastritis"; "enteritis"; diarrhœa; the spleen becomes swollen; a chronic peritonitis develops; **fluid gathers in the peritoneal cavity (ascites),** which may need to be tapped repeatedly, removing anywhere from five to twenty litres at each tapping. There will be also slight obstruction to the bile flow, and the per-

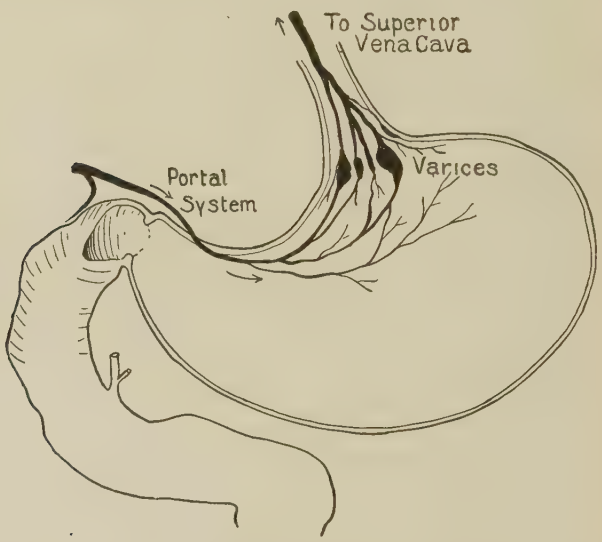


Fig. 89.—Diagram of the stomach showing the venous circulation of the lesser curvature in a severe case of cirrhosis of the liver. The current of blood will now be towards the œsophagus, and the fine anastomoses will be distended into varices. These have thin walls and rupture causing profuse hemorrhages into the stomach.

son will be **slightly jaundiced.** But this is not all. At certain points in the abdomen the portal circulation anastomoses with the general circulation, and through these channels some portal blood can flow to the heart without first passing through the liver. One of these points is the cardiac orifice of the stomach (Fig. 88). The blood from the œsophagus flows directly to the heart, that from the stomach flows to the liver. Veins always anastomose, so that blood here can choose between these two paths. If the portal circulation

is dammed back, a great deal of the blood in the stomach wall will escape through the œsophageal veins; but these veins become distended, forming **œsophageal varices** (Fig. 89), whose thin walls very often burst. Hence patients with cirrhosis of the liver have terrible, often fatal, hæmorrhages from the stomach. Also in the lower rectum is a point where portal and general circulation meet, and, in cirrhosis of the liver, venous varices form here also, which are known as hæmorrhoids or piles, and these too may rupture and cause severe hæmorrhage. Of course **hæmorrhoids** are very common, but in the majority of cases these are due to simple constipation. Such involve the external hæmorrhoidal veins, and are called external piles. These external piles soon become full of clotted blood. But in cirrhosis of the liver it is the internal hæmorrhoidal veins which dilate, and in these the blood does not clot, and so, when these rupture, they bleed badly. Another point where the blood has the choice of two routes is at the navel. There are veins which run from the portal vein through the round ligament of the liver to the umbilicus, there to anastomose with the veins of the abdominal wall. In cases of portal obstruction, a large amount of blood escapes the portal route here also, resulting in a skein of tortuous veins under the skin. These radiate from the umbilicus, forming the "caput Medusæ." And, finally, some of the portal branches unite with the vessels at the base of the mesentery and so escape the hepatic route by flowing into the veins of the posterior abdominal wall.

Through all these anastomoses a compensatory circulation is established, through which enough of the portal blood may get to the heart without passing through the liver to prevent a serious damming back in the abdominal viscera. Surgeons try to imitate this process—try to open up new channels from the outer surface of the liver to the abdominal wall and omentum—by scraping these surfaces so that adhesions will form. The adhesions will contain fine blood-vessels. The results as yet have not been satisfactory.

The chief signs of cirrhosis of the liver are, therefore, a

small liver which can be felt to be too hard, since one feels it distinctly in the midline where no normal liver can be felt; jaundice; ascites; distended venules at the points mentioned in the preceding paragraphs, due to obstruction of the portal circulation; and a flushed face. The severest cases suffer also from toxic symptoms evidently due to the lack of liver function, but closer than that we cannot yet go. That is, the patient has malaise, headache, depression, and later becomes delirious, then comatose, and finally dies in convulsions.

The outlook is bad in all cases of hepatic cirrhosis unless a compensatory circulation can develop adequate to relieve the congestion. It is especially bad if ascites has developed, although there are patients who have been tapped, first frequently, and then less often, until finally a compensatory circulation sets up, and who for years later have become practically well. There is always danger, however, that one of these much distended veins may rupture.

The medical treatment of atrophic cirrhosis includes entire abstinence from alcohol, but also from all highly seasoned foods. A proper diet would contain milk, eggs, fruit, only a little meat, and very little fatty foods, but large amounts of water. Active purgation, especially with salines, is desirable. A gastric hæmorrhage is followed by rest in bed with an ice bag on the stomach, no food by mouth for at least three days, with all water administered by rectum or intravenously (glucose in normal salt solution), and morphia sufficient for quiet. Ascites should be met, first, by purging with salines and diuretics, dimethylxanthin (theocin), and theobromine sodiosalicylate (diuretin), and, if necessary, by tapping.

The hypertrophic cirrhosis of Hanot is a very rare disease, of young men, which is quite a different disease from the above. The liver is very much larger than normal, in fact it may weigh from 2000-4000 Gm. It contains an enormous amount of new scar tissue, but this, instead of being in bands, as in the atrophic form, is quite uniformly

scattered throughout the whole organ. As a result, the liver is large, smooth, and very firm indeed. The spleen is large. There is much less obstruction to the portal circulation, but more to the bile ducts, and the toxic symptoms are worse than in the atrophic form. These cases are marked by occasional attacks of fever, leucocytosis, pain, and enlargement of the liver and spleen, all suggesting infection. No cause can be assigned; we know it is not due to alcohol. It is a very chronic disease, lasting from four to ten years. The jaundice is slight; there is some pain in the liver; some nausea and vomiting, and a rather marked tendency to hæmorrhage under the skin and from the mucous membranes; there is no ascites, since there is no portal obstruction, but the cases usually end as a terminal fatal cachexia with intense jaundice.

Acute yellow atrophy of the liver is due to an acute necrosis of the liver tissue, because of which the liver rapidly shrinks to less than half its normal size, and becomes soft and flabby. This occurs as a rare complication of several acute infectious diseases (especially lues, diphtheria, and typhoid fever), but more often as a result of chloroform, arsenic (arsphenamine), and phosphorus poisoning, and as a complication of pregnancy. It begins as a rapidly deepening jaundice, with intense toxæmia, headaches, vomiting, often of blood, a rapid diminution of the liver dullness, hæmorrhages under the skin, collapse, delirium, convulsions, and death. The urine contains too little urea (since the liver is not functioning), and much of those substances which come from the breaking down of the liver tissue (leucin, tryosin), etc. A few cases have recovered with, as a result, cirrhotic livers.

Abscess. Since the liver is a sieve through which flows all the blood from the digestive organs, it is not surprising that **abscess of the liver** should follow infections of the gastro-intestinal canal. The intestine is always full of bacteria, most of them harmless, but some virulent, and all waiting for a chance to gain a foothold and make trouble. Whenever an ulcer forms in the stomach or intestine, the germs

can get into the blood stream. If an abscess develops anywhere along the gastro-intestinal tract, or in the pelvis, there is always danger that others will form in the liver. Often, the "portal of entry" cannot be found. It is, therefore, little wonder that the liver is so often the seat of infection. The great majority of these bacteria are doubtless killed at once by the blood-plasma, but some get a foothold in the liver and multiply there. The abscess starts in the branch of the portal vein itself in which the infected embolus lodged. The toxins of these germs kill the liver cells in their neighborhood, and this dead tissue seems a protective wall for the germs. Meanwhile, leucocytes migrate from the blood-vessels into the infected area to aid in the protection of the body. The result is soon an abscess cavity full of a liquid consisting of living and dead pus-cells, of liquefied liver cells, and bacteria. Open this cavity, and this liquid "pus" will flow out, leaving an empty hole.

These abscesses may be due to several kinds of germs, but the most common one is not a bacterium, but a protozoön, entamœba histolytica, the most important cause of dysentery. Sometimes these protozoa cause multiple liver abscesses, but more commonly only one, often a large one, even as large as a man's head.

A large amœbic abscess may remain undiscovered for a long time. The symptoms of liver abscess are, in general, fever, pains, and enlargement of, and pains in, the liver. The skin soon becomes pale and jaundiced. The temperature is very intermittent with daily chills and profuse sweats which simulate those of malaria. The poison forming in the liver and carried around the body in the blood gives rise to a general toxæmia. While the abscesses are small, nothing may suggest that the liver is the seat of disease; but soon this organ swells and becomes painful. The body handles the problem by forming a dense wall of scar tissue around an abscess. In this way its advance is often checked, the temperature no longer rises, the pain ceases, the patient feels well and considers himself well; but the abscess is still there

and may at any time cause trouble. As a rule, however, the abscess is not checked, but extends in some one direction and finds an outlet—"comes to a head"—and it is a matter of vital importance where the "head" is. Where the inflam-

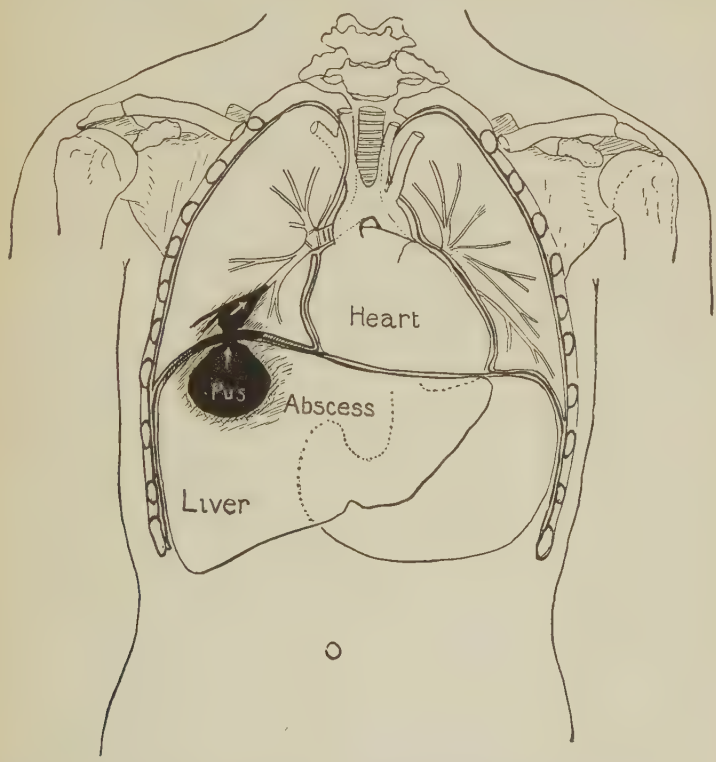


FIG. 90.—Diagram of an abscess of the liver perforating through the lung into a bronchus. The preceding inflammation will have obliterated the peritoneal and pleural cavities in the path of the abscess.

mation reaches the surface, there will be some pleurisy or peritonitis. Fortunately, the abscess often extends upwards. The zone of advancing inflammation always extends ahead of the actual abscess and soon reaches the upper and outer surface of the liver. This at once becomes adherent to the diaphragm, which also becomes inflamed. Since two inflamed

surfaces which touch each other are soon stuck tightly together, liver and diaphragm are now one piece. Then the inflammation extends through the diaphragm, and soon we have a pleurisy above it, as a result of which the lung pleura, and the diaphragmatic pleura become adherent. Soon liver, diaphragm, and lungs, are practically one solid piece. The abscess formation has meanwhile followed the inflammation, and a hole is soon eaten through into the lung. If the union of the liver, diaphragm, and lung is tight, the pus will not pour out into either peritoneal or pleural cavity, but will discharge into a bronchial tube (Fig. 90). In this way it finds a vent, and the patient soon begins to cough up all this liquid pus. If successful, he will empty the abscess, leaving a clean hole. The wall of scar tissue will now get stronger and contract, until some day the hole will all be gone, only a scar will remain, and the patient will be well.

But the abscess may extend to a side of the liver or in a downward direction. Then the same process occurs. The liver becomes united by adhesion of inflammation to the stomach, to the intestine, or even to the wall of the abdomen; and then the abscess will perforate into one of these organs or even make a hole through the abdominal wall and come to a head in the skin. Sometimes nature does not succeed so well, and the pus finds free vent into the pleural cavity, causing empyema; or into the abdominal cavity, causing peritonitis and death.

The treatment is operation. If he is quite sure there is a large pocket of pus in the liver, the surgeon should provide it with a hole through which it can escape freely and safely.

When a gall-stone gets caught in a gall-duct, it causes at once an inflammation, which may extend into the smaller gall-ducts. Each duct may be filled with pus, that is, it is really an abscess. This condition is called **cholangeitis.** Sometimes such abscesses are in the portal veins themselves, (nearly all abscesses begin this way but most remain local) and extend along these throughout the liver, a condition known as **pylephlebitis.**

Cancer. Rarely do **cancers originate in the liver,** and then their starting-point is usually in the bile ducts; but secondary cancers in the liver are very common indeed. The primary growth may be almost anywhere, and since the blood stream and the lymphatics from the body cavities nearly all reach the liver or the diaphragm, which is intimately connected with the liver, malignant tumors anywhere in the trunk are quite certain to reach this organ sooner or later. Moreover, the liver furnishes an ideal place for these cancer nodules to grow. Often it is studded by nodules of such

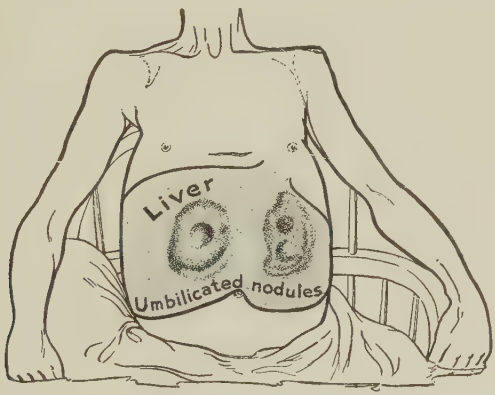


Fig. 91.—Large liver with palpable umbilicated nodules of cancer.

size that the liver, instead of weighing about three pounds, may weigh from ten even to forty pounds. Formerly it was supposed that the liver, acting in its capacity as a filter, filtered loose cancer cells from the blood. While this may be true in part of all malignant tumors, and especially of the sarcomas, it would seem as though the carcinomas, or cancers, grow also by direct extension as microscopic threads along the lymph vessels, which connect the original cancer with some of its metastases. This discovery really brings with it considerable hope, for we are more confident now than ever before of the possibility of curing cancers if we can only operate early and extensively enough. If loose cells

early were sown like seed by the blood stream over the whole body in the way in which infection is spread, there would indeed be little hope.

Often the first evidence of a cancer in an abdominal organ is the appearance of one of its **liver metastases,** and, unless exploratory operation, or an autopsy, is performed, we may never discover the primary growth. One may diagnose cancer of the liver, regardless of where the primary tumor may be, when there is a recently beginning loss of weight, loss of strength, and secondary anæmia (the most common early symptoms of cancer anywhere in the abdomen), together with the rapid enlargement of the liver and the appearance of one or more palpable nodules, which may be umbilicated, on its surface. This is the best illustration of the **large irregular liver.** Jaundice is present only if the larger ducts are closed by the pressure of the cancer nodules in the hilum of the liver, and ascites only if these same nodules obstruct the portal veins. There is no definite treatment.

CHAPTER XII

Diseases of the Pancreas

The pancreas, or the "stomach sweetbread" as it is called in animals, is situated behind the stomach. Its main duct (duct of Wirsung), together with the common bile duct, opens into the duodenum through the ampulla of Vater (Fig. 93). This gland has at least two functions: it manufactures the pancreatic juice, its external secretion, which

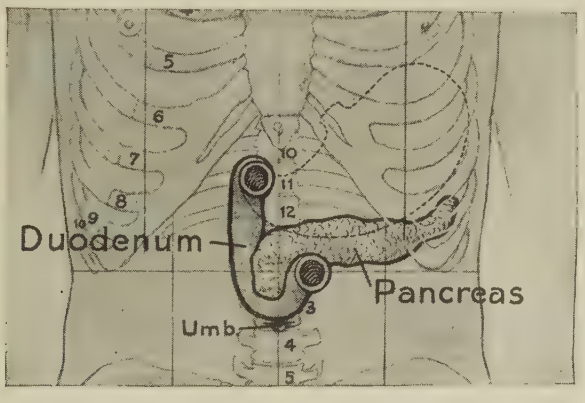


FIG. 92.—Pancreas and duodenum.

has already been described on page 201: and it secretes a very important internal secretion which it pours into the blood stream.

This **internal secretion**—a remarkable product—is entirely distinct from the external secretion. If the pancreas is entirely destroyed by disease, or entirely removed, or suffers certain diseases less evident on inspection, the tissues of our body cannot burn grape sugar, their ordinary fuel. This accumulates in the blood, and the kidneys get rid of it by excreting it in the urine. But if, in the case of animals, the surgeon removes the entire pancreas from its normal position

but grafts a piece of it to any other part of the body, under the skin, for example, the internal secretion of this transplanted piece of the organ will save the animal from diabetes.

Hæmorrhage into the pancreas must be suspected in cases of very sudden death of persons who, apparently in perfect health, die suddenly and without evident cause. Other apparently well persons complain of a sudden agonizing pain

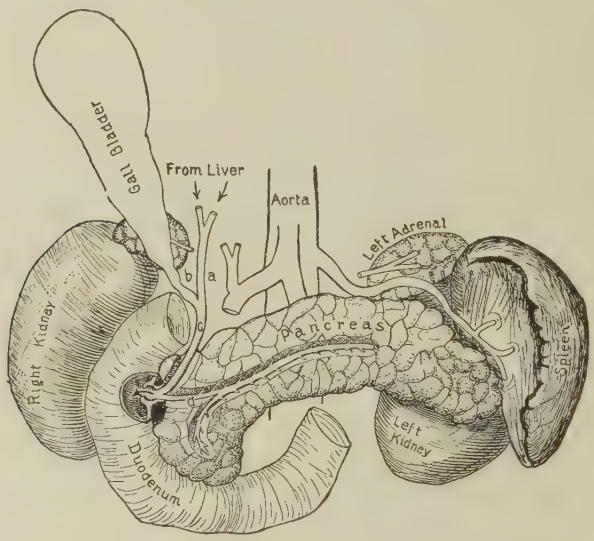


FIG. 93.—Diagram of some of the abdominal organs. The stomach and the liver, which normally cover these organs, are not represented. *a*, hepatic duct; *b*, cystic duct; *c*, common duct. The arrow is in the ampulla of Vater. *d*, pancreatic duct, which is represented as exposed.

in the upper abdomen, and this is followed by collapse, and severe retching and vomiting. The nausea and vomiting may be terrific in severity. Death may follow within a day, or the patient may live two or three days in great agony, with all the symptoms of intestinal obstruction, including fæcal vomiting, and with a firm mass, easily felt, in the epigastrium. In the case of those who die suddenly the diagnosis of heart disease is usually made when no autopsy is performed, and in the great majority of cases this is correct; but in the case

of a very few the pancreas is, at autopsy, found soaked in blood, which may also fill the abdominal cavity. In the case of those who live a few days or so, one finds, in addition to such a hæmorrhage, but not always present, an active inflammation of the pancreas; and one finds in the fat of the abdomen, of the abdominal walls, and even of the chest, **little areas of fatty necrosis.** If death is not quite so rapid, part, or even the whole of the pancreas, may be dead. If the patient lives a little longer than a few days after the attack, an abscess is usually found in and around the pancreas, which may float, a mass of dead tissue, in the pus. All these conditions seem stages of the same process, one differing from the others according to its duration. The rapidly fatal cases are called **hæmorrhage into the pancreas,** or **apoplexy of the pancreas;** those which live a day or so have **acute hæmorrhagic pancreatitis;** the next stage is **necrosis of the pancreas;** the last, **abscess of the pancreas.** Some of the cases, slighter still, develop later a **pancreatic cyst.** Some of the acute cases get well, but the vast majority do not, unless saved by operation.

We know something of the **cause of these conditions.** As is said on page 201, the trypsin of the pancreatic juice is the most powerful ferment known. This is formed as harmless trypsinogen, which cannot digest anything, but which in the duodenum is "activated," or changed, to active trypsin. The change is made by a ferment, enterokinase (page 201), supplied by the walls of the duodenum. The walls of the bowel are able to withstand trypsin; but suppose that by accident a little of the trypsinogen is activated while in the pancreas. That organ cannot withstand its powerful action and is quickly digested in spots; the blood vessels' walls are eroded, and hæmorrhages follow. The trypsin then diffuses freely in the body, digesting the fat in spots. Lipase, also a ferment of the pancreatic juice, spreads with the trypsin, and splits the fat of these digested masses to glycerin and soap, just as it is split in the intestines, when ferments (lipase and trypsin) act on it normally. The areas of dead pancreas are

now an excellent place for germs to grow in, so that inflammation and abscess are the result. Moreover, trypsin injures not only through its digestive action, but is also a powerful, perhaps a fatal, poison.

The treatment for these conditions is to operate, slitting the pancreas open from end to end, and allowing all the fluid or pus to escape.

The reason why trypsin is thus prematurely activated is sometimes evident. Bile can activate this ferment, and has a chance to do this when a gall-stone plugs the common opening of both bile and pancreatic ducts, for then the superior force of the bile current forces bile back into the pancreatic duct. In more cases, however, the bacteria which gain entrance to the duct or gland and the pus-cells which collect where there are bacteria, activate the trypsin.

Pancreatic stones, the stones which form in the pancreatic duct, are composed of calcium carbonate, or lime. The pancreatic juice is rich in this mineral, which in the intestine helps to neutralize the acid from the stomach, and which in fat digestion helps to form soap.

Pancreatic stones can cause a good deal of pain in the upper abdomen, worse after meals, and this pain is usually ascribed to gall-stones, although it usually radiates to the left side. If a stone completely plugs the pancreatic duct, the pancreatic fluid is dammed back and the pancreas begins to atrophy—that is, to shrivel up because of the disappearance of all those cells which furnish the pancreatic juice.

Chronic interstitial pancreatitis resembles cirrhosis of the liver in that the real pancreas tissue disappears, and its place is taken by scar tissue. In moderate grades the organ is large and hard, but in some cases all that is left is a small mass of scar tissue. This may be the result of a permanent obstruction of the pancreatic duct, or of long continued mild infection by the germs which inhabit the gall-ducts in nearly all gall-stone cases. The result is a slow diminution of the external secretion of the pancreatic juice, but not necessarily

of the internal secretion of the pancreas, which, even in advanced cases, is spared.

When, because of disease of the pancreas, or obstruction to its duct, no pancreatic juice at all is furnished the intestine, there may for a time be no symptoms. The gastric and intestinal juices can do some of the work of the pancreatic juice without the digestion suffering. **The chief difference early will be in fat digestion,** for less than one fifth of the fat will be used, and the rest will appear in the stools. Later, however, the effect of the loss of the pancreatic juice becomes more apparent. **The diarrhœa pancreatica begins.** That is, the patient four or five times a day will pass stools which are huge in size, because so little of the meat (even visible in the stools) and fats which are eaten is digested, and because they contain much gas, the result of fermentation. As so little food is digested, the patient will soon be very thin, no matter how much he eats, and will slowly starve to death. Later begins the "fatty diarrhœa." The fat, no longer split, is passed in a state like liquid oil or melted butter, either with the fæcal matter, or alone, and may continually ooze from the rectum. It quickly hardens as the stool cools.

If the pancreas is so injured by those conditions described that too little of its internal secretion is formed, then the **patient becomes diabetic,** that is, cannot use glucose properly, and, as a result, this sugar is eliminated in the urine (diabetes mellitus, page 369). But to produce this condition, the visible destruction of the pancreas must be complete, for atrophy sufficient to stop all external secretion may not seriously affect the internal secretion. Since, in the majority of cases of true diabetes mellitus, the pancreas, so far as one can tell by superficial inspection, is perfectly normal, surely the essential cause of diabetes is not total destruction of this organ. The internal secretion of the pancreas is not formed by the same tissue as the external, but by little areas scattered throughout its substances, called "islands of Langerhans," and destruction of that part of the pancreas which

furnishes the external secretion does not always destroy these islands. Some forms of pancreatitis can be seen to injure these islands without injuring the rest of the pancreas, but in more cases of diabetes it is not evident just what is the trouble with these islands.

Pancreatic cysts usually appear as a bulging of the upper, rarely lower, abdomen in the midline. These masses do not move on respiration, nor can one move them on palpation. They may grow to large size, practically filling the abdomen.

Some pancreatic cysts, which follow injury to the upper abdomen, start with pain, vomiting, and signs of inflammation. Some have an onset suggesting gall-stone colic. Others begin quietly and may last for over twenty years. The symptoms will be those of direct pressure on the neighboring organs, colicky attacks, nausea and vomiting, jaundice, gastric and intestinal disturbances, and dyspnœa. In addition are those symptoms due to the injury which the pancreas itself has suffered. If, in the formation of the cyst, the most of the pancreas is destroyed, then loss of weight, fatty stools, and glycosuria will develop. The treatment is surgical removal of the tumor if possible, and drainage if not possible.

Cancers of the pancreas are not rare. These usually are first indicated by an intense permanent jaundice, (since the common bile duct must almost pass through the head of the pancreas before it enters the duodenum); sometimes a severe boring pain which extends through to the back; dilatation of the gall-bladder; loss of weight and strength; and all the symptoms of deficient pancreas function (see page 251).

Treatment can be only symptomatic, although the permanent draining of the gall-bladder may relieve the symptoms due to the jaundice.

CHAPTER XIII

Diseases of the Kidneys

The Organs of Elimination

In Chapter VIII we discussed the general subject of foods, their nature, the processes of their digestion, and the uses the body makes of them. Finally, all of the food absorbed from the gastrointestinal tract is burned, sooner or later, and in this combustion ashes are formed which must be removed from the body just as regularly and completely as ashes must be removed from a furnace. In the present chapter we are to study **the organs which remove these ashes.** But the ashes as they leave the cells—the furnaces—are not the same as those found in the urine. The ashes of proteins, for illustration, are water, carbon-dioxide gas, and a number of very simple ammonia bodies, of which we know little except that they are distinctly poisonous. Lest these injure us, the "ash heap" must be "worked over." This is done by the liver, perhaps the muscles and other organs (see page 54). They remove from the blood these **ammonia ashes and build them over** into the non-poisonous substances, **especially urea,** which we find in the urine. The ashes of protein when excreted are, especially, water, carbon-dioxide gas and urea, but also uric acid, creatinin, xanthin, hypoxanthin, adenin, hippuric acid, and a number of other nitrogenous bodies—some known, others not yet isolated; also chlorides, sulphates, phosphates, and other mineral salts.

The ashes of fats are water and carbon dioxide. The ashes of carbohydrates are the same.

Water is already an ash (of hydrogen) and hence cannot be burned, but is taken, used, and eliminated as water. The same can be said of most of the mineral salts (e.g., table salt, or sodium chloride).

The organs of excretion which free our body from these ashes are the kidneys, lungs, intestines, liver, and skin. **The lungs** excrete the most of the carbon-dioxide gas and about

one fifth of the water. **The skin** eliminates some of the water and a very small part of the salts. In Bright's disease it may get rid also of a little of the nitrogenous waste, for, when a sufferer from this disease is near death, urea sometimes crystallizes out as "urea-frost" on the skin, forming little solid masses about the size of the head of a pin, thickly clustered over the skin of the face. It is certain, however, that this excretory power of the skin is negligible, and that the real value of a sweat bath is its effect on the distribution of the blood.

The intestine also is an excretory organ. We do not refer to the excrementa, the refuse which could not be digested or absorbed and which merely passes through the intestine, but to some salts and ashes which the liver and the wall of the bowel remove from the blood. Of the salts, those of lime are, for the most part, excreted by the bowel wall. When the kidneys are in trouble the intestine can do some of their work, and so a patient with Bright's disease is purged that the bowels may help still more. The liver also is an excretory organ, for the bile contains a considerable waste (page 231).

The Normal Urine

The kidneys get rid of most of the ashes of the body as a solution we call urine. In the urine are excreted about four fifths of the water to be eliminated, a little of the carbon dioxide, and by far the most of the solid ashes and salts—all but the little which the skin and bowels remove. In the urine also are to be found nearly all of the poisons and foreign soluble bodies which by accident have got into the blood.

Urea deserves special mention. In pure condition, it is in beautiful white crystals which are very soluble in water. Urea is not, as stated above, an original ash, but a non-poisonous modification of the ammonia ashes (page 257). Urea is not at all poisonous, and yet the condition uræmia, a severe auto-intoxication (page 280), was, because of an erroneous theory, named after it. Urea is composed of nitrogen, carbon, hydrogen, and oxygen, and about one half of it by weight is nitrogen. Nearly ninety per cent of the total nitro-

gen eliminated from the body is in urea, and since it is very much easier to determine the amounts of urea than of the nitrogen in the urine, doctors for years estimated only the former. A normal person voids between twenty and thirty grammes (almost an ounce) of urea every day, and every gramme represents three grammes of protein which have been burned. A person on a poor diet may void from but fifteen to twenty grammes, while one on a very rich diet may void as much as one hundred grammes of urea a day. The amount of urea in the urine depends on two things; the amount there is in the blood to be excreted, and the ability of the kidneys to get rid of it.

Normally about 5 per cent of the nitrogen of the urine is in the form of **ammonia salts.** This ammonia is some of that which was not transformed into urea, since it previously had been used as an alkali to neutralize acids formed in the body.

Uric acid is often seen in clear acid urine which has stood for a while (especially if a little hydrochloric acid has been added) as masses of brown or red crystals which resemble red pepper. When purified from this coloring matter uric acid crystallizes out in white crystals. It (together with xanthin, hypoxanthin, etc.) is quite certainly an ash of the nuclei of cells. A little less than one gramme is present each day in the urine. No substance in our bodies, perhaps, has been the subject of quite so much romance as this acid. Forty-six different diseases have been attributed to it, and this list includes gout, rheumatism, nervous disorders, and even bad vision. That it is retained in the body in gout is granted, but its importance in all other diseases is questioned. In reptiles the major part of the nitrogen eliminated from the body is in the form of uric acid rather than urea.

Creatinin is an important substance since, next to urea, it is one of the most abundant of nitrogenous excreta (1 to 2 grains each day). There are fifteen or twenty other known nitrogenous substances always present in the urine, perhaps five grammes in all each day, none of which, as yet, has much interest for us.

Of the **salts**, sodium chloride is the most important, and of this the urine contains about fifteen grammes (one half ounce) each day. Of sulphates, it contains perhaps five grammes, of phosphates, perhaps two, and of many other salts only traces —their number and amounts depending, of course, on the food we eat.

Sugar (glucose) is present in traces in all normal urines, perhaps 1 Gm. per litre, but not enough to be detected by ordinary tests. In several conditions, however, much larger amounts are present, the most important of which condition is diabetes mellitus. In this disease the kidneys are not diseased, but act as a safety-valve allowing, for the good of the body, any excess of glucose of the blood (over 160 mg. per 100 c.c.s.) to escape in the urine. In various diseases of the nervous system also, e.g. after injuries to the head, there may for a brief time be moderate amounts of sugar in the urine.

The Benedict test is, perhaps, for the use of nurses, the best test for sugar in the urine (Joslin). It is easy to use, it is delicate (some say too delicate since it detects quantities as small as 0.1 per cent), and the solution used will keep indefinitely.

The formula of the Benedict solution is:

	Grammes or c.c.
Copper sulphate (pure crystallized)	17.3
Sodium or potassium citrate	173.0
Sodium carbonate (anhydrous)	100.0
Distilled water, to make	1000.0

The citrate and carbonate are dissolved together (with the aid of heat) in about 700 c.c. of water. This solution is then poured (through a filter if necessary) into a larger beaker or casserole, and the copper sulphate (previously dissolved in about 100 c.c. of water) then slowly added with constant stirring. The mixture is then cooled and diluted to 1 litre.

To 7 c.c. (an ordinary teaspoon holds about 5 c.c.) of Benedict's solution measured into a test-tube are added from an unbroken medicine dropper eight (not more) drops of

the urine to be examined. The tube is agitated to mix the urine and the solution, and is then placed in bubbling water where it must remain, with the water actually boiling, for five minutes. It is then removed and shaken. (The discoloration which occasionally forms upon the surface is unimportant and disappears with shaking.) If the solution is clear (no precipitate), the urine tested is sugar free; if it is slightly cloudy, but one can still read print through it, the amount of sugar present is so slight that it can be disregarded; if a heavy greenish precipitate forms it usually means that a trace of sugar is present; a yellow sediment means that less than one half per cent, and a red sediment, that more than this is present; while if the supernatant fluid, after the precipitate has settled, is water-clear, the urine contains over 1.5 per cent of glucose.

Albumin. Normal renal cells allow a trace of albumin to pass into the urine, but this trace is so minute that it cannot be detected by the ordinary tests. If any at all can be recognized by these tests an albuminuria is present and something is wrong. It may not be anything serious, and the condition producing this albuminuria may be very temporary, yet, in the majority of cases, the cause of a long standing albuminuria is nephritis (called also Bright's disease). On the other hand the kidney may be the seat of a severe nephritis without any albumin appearing in the urine.

The test for albumin is made as follows:—a clean test tube is filled three quarters full of perfectly clear urine; holding the tube at its lower end, one heats to the boiling point the upper one half of the column of urine in a gas or alcohol flame. The flame should not be allowed to strike the glass above the level of the fluid, and the tube is constantly shaken a little in order to prevent breaking the glass. As soon as the boiling point is reached, one adds two or three drops of 5 per cent acetic acid and then again heats the same portion of the fluid to the boiling point. Then, holding the tube against a dark background, one compares the clearness of the boiled and unboiled halves of the column of urine to detect

any cloudiness in the heated portion of the urine. If the heated portion is cloudy, albumin is present. The cloud which often appears after the first boiling, but which disappears when the acid is added, is a phosphate precipitate which means nothing. Since the cloud indicating small amounts of albumin does not appear at once, the tube, after the test described above has been completed, is **allowed to stand in a test tube rack for at least five minutes** and the test is not pronounced negative unless the cooled urine remains quite clear after that period of time.

There are some persons who for years have at times albumin in their urine. Some of these, usually thin, weak appearing individuals, have an **albuminuria only while in the erect posture;** the urine they secrete while lying down contains none. The cause of this so-called **orthostatic albuminuria** resides in the circulation of the kidney rather than in any disease of this organ. Some of these patients have a very low pulse pressure; others, a spine so curved forward (lordosis) as to interfere with the renal circulation. The therapy necessary for these weak young persons is any physical training, diet, tonic, etc. which will make them stronger. It should be remembered, however, that any albuminuria, whatever its cause, is always more or less orthostatic; that is, that in Bright's disease, for example, the albuminuria tends to increase when the patient sits up and to decrease when he lies down, and that while a case of Bright's disease is recovering the last traces of albumin appear only while the patient is erect.

Another group of persons with albuminuria which does not indicate Bright's disease includes those who **do anything excessively.** Many strong, well trained persons as foot-racers, foot-ball players, soldiers on forced marches, et al, have albuminuria at the end of the game or race. This is said to be true also of swimmers who remain **too long in cold water,** of persons who overeat, and of some individuals, it is claimed, after a period of intense mental exertion. The albuminuria in all such cases is temporary, its degree is slight,

and its duration ephemeral. These persons, it is assumed, have perfectly healthy kidneys, and yet that is a point very difficult to prove. A few of them at least may for years have had an unsuspected nephritis, but one so very slight that the albuminuria appears only after some unusual strain.

Some young persons who, it is claimed, belong to neither of the above groups, may for years have a slight constant albuminuria. In many of them, the condition later disappears and the patient remains well; others are never in the best of health. These persons have what is called a **functional albuminuria,** because one assumes the cause is not nephritis. This may be true of those with an undiscovered heart trouble serious enough to cause chronic passive renal congestion (page 275), but the majority of this group had, during childhood, scarlet fever, measles, diphtheria, or tonsillitis, etc., from which they seemed to recover perfectly but which left behind a slight permanent nephritis.

Many cases of acute fever have, while the temperature is elevated, a so-called **febrile albuminuria** which disappears with the fever. In these cases, the kidneys are not normal.

The Urine in Renal Disease

Most cases of albuminuria, finally, are **due to true Bright's disease**; that is, to **nephritis.** The amount of albumin in these cases varies enormously. In the acute cases, much is present, so much even, that when the urine is boiled it truly solidifies. The more chronic cases have less, and the very chronic cases least—so little that only an expert can find it, and even that trace may for weeks at a time be absent. These chronic cases, however, may have, during an acute exacerbation of their condition, as much albumin in their urine as have cases of true acute nephritis. One thing is certain; **from the amount of albumin** one can never judge **the extent of the permanent renal injury.** A case of acute nephritis which later recovers may have maximal amounts; a case dying with severe chronic nephritis may have only a trace.

Casts. One hears much about the casts (or cylinders) in the urine. These get their name from the fact that they owe

their pattern to the renal tubules which serve as their mould. That is, the tubules become filled at points with some substance which hardens and thus forms a "cast" of their lumen. These casts are later washed out by the urine where they can be found with a microscope. A portion of the epithelial lining of a tubule which becomes detached and appears in the urine as a short cylinder lined by epithelial cells is called an epithelial cast. The name epithelial cast is given also to masses of renal cells which became detached

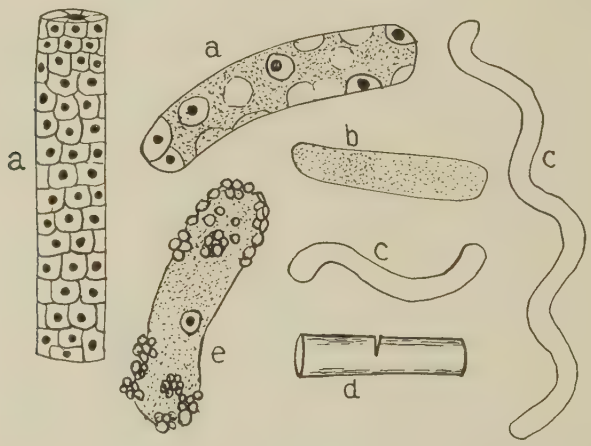


Fig. 94.—Casts found in the urine. *a*, epithelial casts; *b*, granular cast; *c*, hyaline casts; *d*, waxy cast; *e*, fatty cast. (Much magnified.)

and glued together within the lumen of a tubule. More often these cells maintain their places in the wall of the tubule, but their free margins become disintegrated, setting free granular detritus which becomes massed in the tubule, forming a **granular** cast. According as these granules are fine or coarse, occur the **finely** and **coarsely granular casts.** Or the cells of epithelial casts may become very fatty, in which case they are called **fatty casts.** The most common casts of all, however, are the **hyaline casts,** composed of a pale translucent material, perhaps from the blood, which hardened in the tubule. These are very hard to see. Others also are very

clear, but easy to see since they look as if made of wax. These are the so-called **waxy casts. Blood casts** and **pus-casts** are terms requiring little explanation. Some are made up of these cells which had become massed together in the tubules, but these names are applied also to any hyaline or granular cast with even one red cell or leucocyte attached.

Casts in the urine (cylindruria) have much the same clinical significance as albuminuria. As a rule albumin and casts appear together, but for short periods either may occur without the other.

14 **Red blood corpuscles** may be found in the urine—either a few, or many, or so very many that the urine looks like blood. When these are sufficient in number to change the color of the urine the condition is called hæmaturia. One practically always finds red blood cells in the urine in acute nephritis and during the acute flare-ups of chronic nephritis. In these cases the urine is not often red but is more "smoky" in appearance. The daily approximate count (estimated under the microscope) of the number of red blood cells in the urine is **one of the best means of determining the severity of a nephritis,** and, if the count is made daily, of determining its progress. The presence of blood cells is one of the best means of distinguishing a **subacute tubular nephritis** (in which they are practically absent) from a **subacute glomerular nephritis** (in which they are numerous). They appear in the urine also as a result of renal congestion due to heart disease, while a true hæmaturia is common in cases of renal stone, of tuberculosis, or of cancer of the kidney, and in all cases with bleeding lesions in the kidney, ureter, prostate, or in the bladder, such as a very acute inflammation, or an ulcer due to a stone, cancer, tuberculosis, or similar cause. In all such cases the source of the blood must be determined by the general condition of the patient, aided by the use of the cystoscope and of the ureteral catheter.

15 Some young and otherwise healthy persons have occasional attacks of what is called **essential hæmaturia,** that is, of spontaneous bleeding from the kidney (always from one

kidney only). Because of a certain superficial similarity these cases have been called "renal epistaxis" (or renal nose bleeds). It would be very difficult to prove that these kidneys are entirely normal and, as a matter of fact, some of them have varicosed veins in their pelvis while others later develop some serious disease. As a rule, however, this tendency to renal hæmorrhage disappears without treatment, leaving the individual quite well.

Finally, **hæmaturia** may be part of some more **general hæmorrhagic disease,** as purpura, scurvy, leukæmia, or the hæmorrhagic types of the infectious fevers, as "black" typhoid, "black" measles, etc.

In all the above cases the treatment will depend on the cause of the hæmaturia.

In the condition called **hæmoglobinuria,** the urine is red or dark brown in color, not from the presence in it of red blood cells, but of free hæmoglobin. The cause of the hæmoglobinuria is an hæmoglobinemia, due to the destruction of so many red cells that the liver cannot properly warehouse the hæmoglobin freed in such quantities, with the result that some remains in the plasma, and this, if sufficient to pass the threshhold level of the kidney, is eliminated in the urine. Hæmoglobinuria is seen in severe malaria, after the ingestion of certain poisons, as phenol, or potassium chlorate, and in severe cases of many infectious diseases. **Paroxysmal hæmoglobinuria,** a late result of lues, is a condition marked by sudden attacks of hæmoglobinuria which follow exposure to cold of even a small area of the body. The urine during the attack has a "black" (dark burgundy red) color. These attacks, which may begin in a few minutes after the exposure or several hours later, are ushered in by malaise, pains in head, abdomen, or legs, chills and fever, and cyanosis. The treatment is that for lues.

Pus-cells can always be found in the urine in cases of nephritis or any other inflammatory conditions. When present in sufficient number to give the fresh urine a cloudy appearance the condition is called **pyuria.** Pyuria occurs in

pyelitis, in tuberculosis of the kidney, and, especially, in cystitis and urethritis.

Epithelial cells always are present in the urine. One might expect this since these cells are constantly being shed from the surfaces of the ureters, bladder, and urethra, as from all epithelial surfaces. Their number is greatly multiplied in inflammatory conditions of the urinary tract.

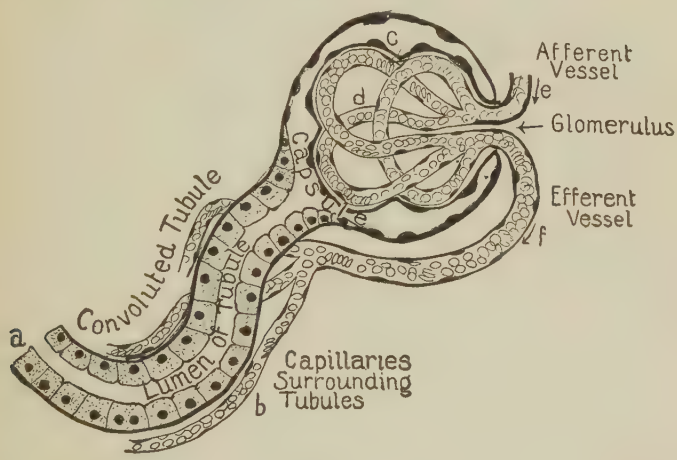


Fig. 95.—Upper end of convoluted tubule together with a glomerulus. *a*, convoluted tubule; *b*, the capillary surrounding *a* tubule; *c*, "Bowman's capsule"; *d*, the capillaries which make up the glomerulus; *e*, the afferent artery which breaks up into the capillaries *d*; *f*, the efferent artery which breaks up into the capillaries *b*. These capillaries *b*, unite to form the renal veins.

The Kidneys

Anatomy. The next question is, **How does the kidney do its work?** How does it separate from the great volume of the blood flowing through it the waste contained therein, and allow nothing of value to the body to escape? The kidneys, like other organs, are made up of little kidneys, and to understand one of these is to understand the whole kidney. Each of these little kidneys is made up of living cells arranged in tubes. Fig. 95, *a*, represents the upper end of a tube. Its wall is made up of these renal epithelial cells, on the outer side of which is a fine network of capillaries, *b*, through which blood is constantly flowing. These little living cells are, there-

fore, constantly bathed on their outer surface by the blood-plasma. They remove from this, by their secretory activities, a very concentrated fluid containing the most of the solid constituents of the urine. This fluid starts to flow along the lumen of the tube towards its open end. At its upper end, this tube swells out into a hollow ball called Bowman's capsule, *c*. At that pole of this ball which is opposite the point where the tube enters, the wall is pushed in, much as a child can push one half of a rubber ball into the other half, by a knot of capillaries, the **glomerulus,** *d*. These glomeruli can just be seen by the naked eye as red dots about the size of a pin prick. The wall of this hollow ball is lined with flat epithelial cells quite unlike those lining the tube. The capillaries of the glomerulus, all of which branched from one vessel, *c*,—a branch of the renal artery—unite again into one vessel, *f*, which leaves the capsule, flows down to the tube, and again breaks up into the network of capillaries, v, mentioned above as surrounding the tube. Through the walls of the capillaries of this glomerulus, pour out into the capsule, by a process quite similar to simple filtration, the most of the water and salts of the urine, and also several other constituents which can pass through a filter. This dilute urine flows down the tube and unites with the very concentrated urine already there. Some of the substances which the glomeruli filtered out are of use to the body and these the convoluted tubules reabsorb back into the blood stream, the selective secretory action of these cells thus correcting the results of the more mechanical actions of the glomerular epithelium. The urine, as thus finally formed, flows down to the open end of the tube into the pelvis of the kidney.

The capillaries surrounding the tubules reunite to form the renal vein, the blood of which now is practically free from waste. A great amount of blood flows through the kidneys, nearly ten times as much as through any other organ of the same weight. In this way the kidney is continuously purifying the entire volume of blood. Just how the cells of the tubules select and separate the waste products from the

plasma, and allow nothing else to escape, is a splendid illustration of the efficiency of our secretory tissues. All the different secretory cells in the organs of our body may look much alike and yet they make no mistakes. They all have the same blood to deal with. The cells of the stomach separate out the materials for gastric juice; those of the pancreas, the constituents of pancreatic juice; those of the liver, bile; and the kidney cells take what the others reject and nothing else. The blood contains large amounts of albumin, some sugar, and many other substances, but the normal renal cell allows little of these to pass. That these renal cells work hard is shown in part by the large amount of food and oxygen which they require. Pinch the renal artery for half a minute, and it is forty-five minutes before these cells start to work again; and when they do, the first urine they secrete shows how injured they were.

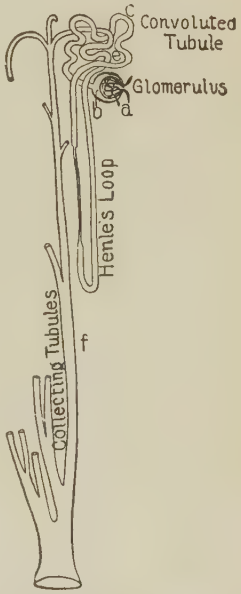


FIG. 96.—The "unit" of the kidney. *a*, **glomerulus**; *b*, capsule; *c*, convoluted tubule; *f*, collecting tubule.

In some very simple animals one such tube as we have described, or a row of them, is all the kidney the animal requires, but for man great numbers are necessary. To economize space, they are not straight but twisted. Their course is much as follows. The tube, when it leaves the capsule (Fig. 96, *b*,) is very tortuous and is called the **convoluted tube,** (Fig. 96, *c*); it then makes a long straight loop, the loop of Henle, (Fig. 97, *d*); then again it is convoluted, *e;* then it opens into a long straight tube, the **collecting tube,** *f*, which grows larger and larger in its course because it receives from all sides many similar tubes. An organ made up of a mass of these tubes, all flowing in the same direction, must of necessity be pyramidal in shape, since the convoluted tubes and the glomeruli are all in the outer layer. In the

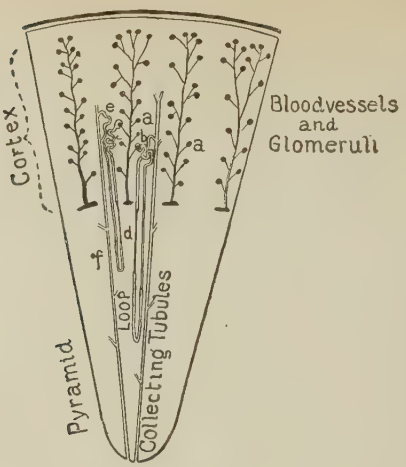


FIG. 97.—Scheme of one pyramid. *a*, the glomeruli arranged like a bunch of grapes on the artery; *b*, convoluted tubules; *d*, loop of Henle; *f*, collecting tubule.

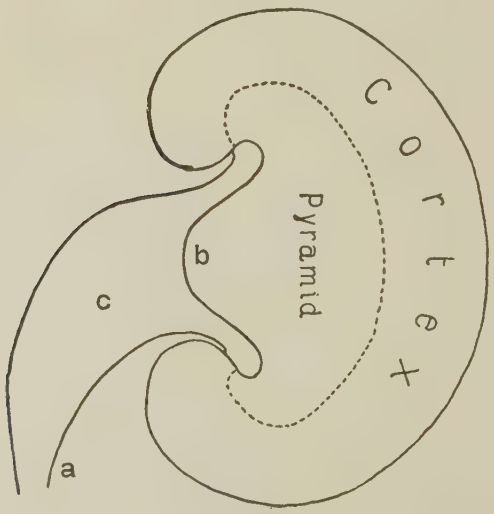


FIG. 98.—A "simple" kidney with only one pyramid. The dog, cat, rabbit, etc., have kidneys something like this. *a*, ureter; *b*, pyramid; *c*, renal pelvis.

dog, for illustration (Fig. 98), the kidney is shaped like a pyramid or cone. In this we can distinguish the outer zone, **the cortex,** where are all the glomeruli and all the convoluted tubules, and the conical end, **the pyramid,** *b,* where are the loops of Henle and the collecting tubules which open on the surface of the pyramid. The cortex is the most important part of the kidney because here are all of its important structures. It is a zone about 4 to 6 mm. in thickness. In it the glomeruli are arranged on little vertical arteries, like bunches of grapes, and between these rows of glomeruli are the convoluted tubes. The tip of the pyramid projects into a dilated sac, **the pelvis,** *c,* which becomes constricted to form **the ureter,** *a,* and this empties into the urinary bladder. The urine therefore flows down the tubes into the pelvis of the kidney, then down the ureter to the bladder.

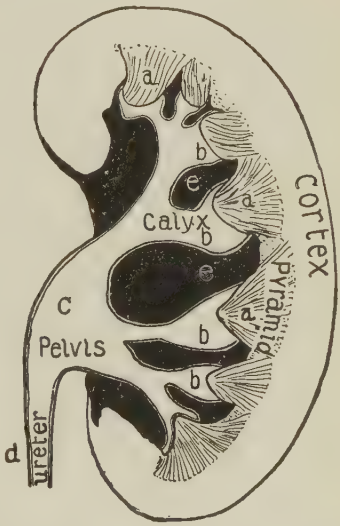


FIG. 99.—Diagram of a human kidney. *a,* pyramid; *b,* calyx; *c,* pelvis of the kidney; *d,* ureter, *e,* space between calyxes, filled with fat.

In man each kidney (Fig. 99) is not a single pyramid but eight or more massed together. In childhood, and in some persons for life, these simple kidneys can be clearly outlined from the lobulation of the cortex. Each pyramid, *a,* has its own pelvis, here called the calyx, *b,* and these calyxes unite to form the pelvis, *c,* which by constriction becomes the ureter, *d.* The intermediate spaces, *e,* are filled with fat.

The two kidneys together weigh about 250 grammes. Sometimes there is but one kidney, or, rather, the two are united into one, a **fused kidney.** Sometimes the two are fused at one pole only, and this, because of its shape, is called a "horse-shoe kidney."

Physiology. A normal person voids about 1000 c.c. of urine a day. **This normal amount,** of course, varies—even from 500 to 3000 c.c. per day (i.e. per twenty-four hours). The quantity depends, first, on the amount of water drunk, and, secondly, on the amount of fluid lost in other ways, as by profuse perspiration, diarrhœa, vomiting, etc. Normally the formation of urine by each kidney is continuous.

Under pathological conditions the amount varies enormously. A large output of urine **(polyuria)** normally follows

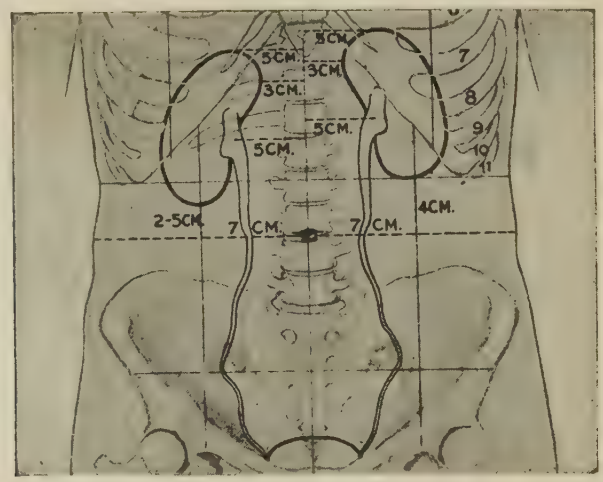


FIG. 100.—Kidneys and ureters (anterior view).

the drinking of large amounts of water, and pathologically occurs in diabetes insipidus (page 275), in diabetes mellitus (page 373), after acute fever (page 275), and in some cases of nephritis. A polyuria is said to be present when the daily output is over 3000 c.c.s. per day. By **anuria** is meant a more or less total suppression of urine. That is, the kidneys produce none or at least less than 50 c.c.s. of urine per day. If the amount is between 50 and 500 c.c.s. the output is said to be pathologically reduced. Anuria will, of course, be the result if disease actually destroys both kidneys, or if some poison, like mercuric bichloride, entirely stops their function;

when there is complete closure of both ureters, as by renal stones or by pressure from outside, as by cancers of the ureter. The closure of one ureter or the removal or destruction of one kidney will as a rule have no influence on the amount of urine secreted since the other kidney, if normal, can easily do the work of both. Sometimes a condition which causes anuria on one side will reflexly stop the action of the other kidney temporarily. Unilateral anuria can be determined only by inserting catheters into both ureters. In cases of bilateral calculi, and in some of the reflex cases mentioned above, an anuria may continue for even nineteen days and then recovery follow. When the cortex is diseased by that inflammation called **nephritis,** the secreting cells are more or less affected and the amount of urine they produce will vary **almost in inverse proportion to the acuteness of the disease** of these cells. In acute nephritis and in acute flareups of chronic nephritis the amount of urine and of its normal constituents is diminished and it contains more or less albumin, casts, pus and blood. In nearly all cases of acute nephritis (except of the very young) chronic lesions also are always present, the results of previous acute processes, and on these in general does the prognosis of the case depend. They, however, only indirectly influence the quality of the urine, but they do influence the quantity. In chronic nephritis, for illustration, during the periods when no acute process is in progress, the urine for weeks may be almost normal in amount and quality. If, however, the amount of cortex has been greatly diminished by disease the urine (when no acute element is in progress) may contain no abnormal constituents but is very dilute and great in amount. A slight superimposed acute process will, however, change all this, and will leave the total injury to the cortex somewhat increased.

The amount of urine secreted by kidneys which are practically normal will in general **vary directly with the amount of blood** which flows through them. This is easy to understand, since the kidney cells can remove the waste from that blood only with which they come in direct contact. In

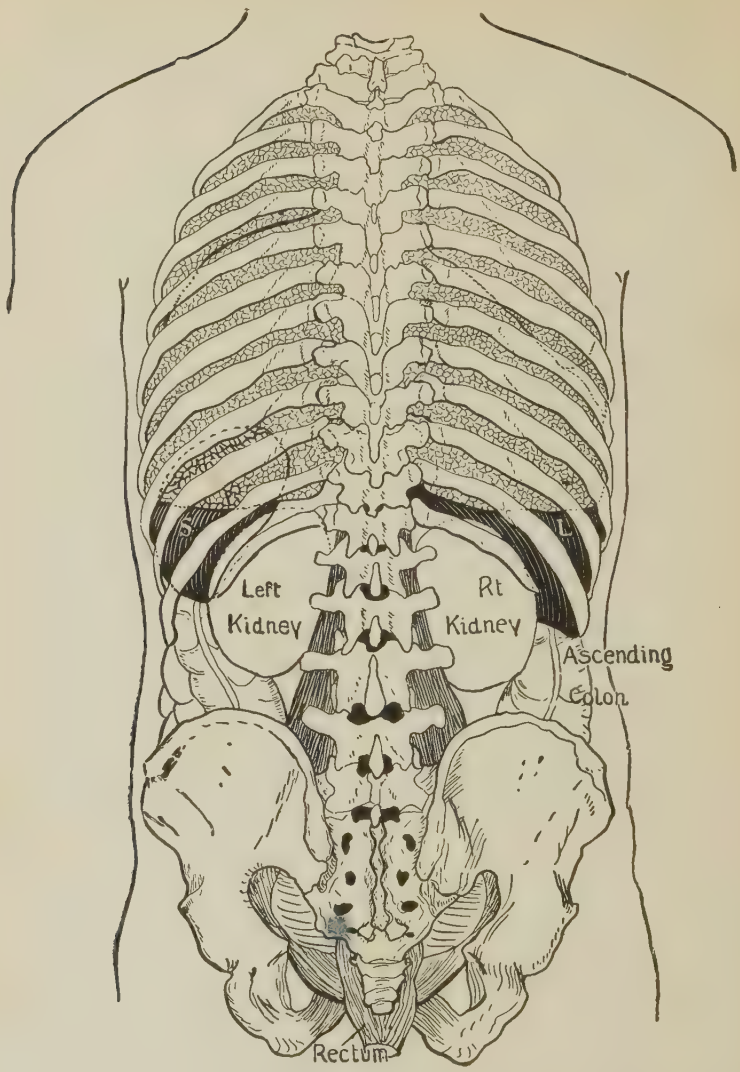


FIG. 101.—The abdominal organs, rear view. *S*, spleen. *L*, liver.

heart-disease with broken compensation, and therefore with the venous blood dammed back into all organs, including the kidneys, it is not strange that the volume of urine excreted should be reduced to a small amount or even to none. In such cases, certain drugs, digitalis for illustration, will greatly increase the output of urine, and therefore serve as diuretics, although they do so not by any direct effect on the kidney, but by helping the heart and thus relieving the renal congestion. The amount of urine depends very little, if at all, on the blood pressure, excepting as this may influence the volume of blood flowing through the kidneys.

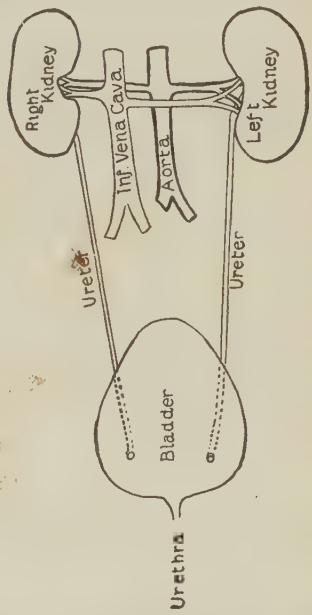


Fig. 102.—Diagram of the urinary organs.

When, however, there is in the blood **an unusually large amount of some substance to be gotten rid of,** the kidneys, in eliminating this, will excrete a large amount of water also to "wash it out." In diabetes mellitus (page 373), for illustration, the patient's urine sometimes contains a pound of sugar a day, and, in order to eliminate this, the kidneys will at the same time excrete from three to even forty litres of water. After acute fevers, typhoid especially, the patient may for a few days void daily from three to twelve litres of urine. The need for this must be the elimination from the body of some substance or substances which had accumulated there during the fever.

Diabetes insipidus is a disease the one symptom of which is an enormous daily output, six or eight, or even 20 litres, of very dilute urine which looks like water, and which has specific gravity from 1001 to 1005. It contains no abnormal sub-

stances as sugar, albumin, etc. The cause of some of these cases is a definite lesion of the brain (tumor, fracture of the skull, lues, etc.) and this, of course should be treated. The disease may begin at birth; in adults it sometimes comes on gradually, sometimes suddenly, after a fright or an injury. The general health is little affected. The so-called primary cases, however, which have no demonstrable cause, and with intense renal congestion (but no disease) as the only demonstrable lesion, are supposed to be due to some cerebral trouble located possibly near the pituitary gland. In these cases it is the polyuria, not the excessive thirst (polydipsia), which is primary. One cannot control the condition by limiting the intake of fluids. Attempt to do this, and the patient will become insane, truly mad for fluid. The hypodermic injection of pituitrin (some inject 1 c.c. three times each day) is the only remedy which diminishes greatly this polyuria. This will reduce the amount of urine voided each day from ten to twelve litres to perhaps three or less, and, as a result, give the patient more restful nights.

In certain nervous conditions, as hysteria, after epileptic convulsions, etc., the person may for one day void a great deal of urine.

Diuretics are drugs which increase the flow of urine. Cardiac diuretics, as digitalis, act on the heart and increase the flow of urine by improving the circulation through the kidneys; others, as caffeine, diuretin (theobromin sodio-salicylate), and theocin (dimethylxanthin), act directly on the renal cells, stimulating them to work harder. Some foods, such as apples, coffee, etc., do this for some persons; certain salts also are diuretic because they are foreign to the blood and so must be eliminated, the kidneys at the same time excreting considerable water. This is why we put potassium salts in lemonade.

The determination of **the specific gravity** of the urine is now one of the nurse's duties. By the specific gravity of urine is meant the weight of a given volume of urine as compared with the weight of the same volume of water. A litre

of water at a certain temperature weighs 1000 Gm., and thus 1000 is taken as the standard of specific gravity. Suppose now you dissolve in water a quantity of urea, sodium chloride, etc. The weight of this solution will be increased, but its volume will change very little if any. Measure now just 1000 c.c. of this fluid. Suppose its weight to be 1015 Gm. Then the specific gravity of that fluid it 1015. When we say "The specific gravity of urine is 1026," we mean that one litre of it will weigh 1026 Gm. More accurately, the "unit" of specific gravity is 1 instead of 1000, and the specific gravity of the fluid just mentioned is 1.026; but in the case of urine it is more customary to write it "1026." We may say, "ten twenty-six," but we never write it 10.26; that would mean that that urine was heavier than iron (sp. gr. 7.7), and almost as heavy as lead (sp. gr. 11.4).

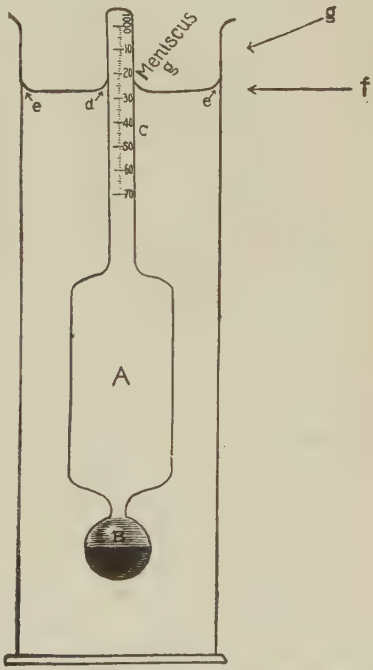


Fig. 103.—Urinometer. *A*, the bobbin. *B*, the weight. *C*, the stem. *d*, the meniscus on the stem; *e*, the meniscus on the walls of the glass vessel; *f*, proper line of vision; *g*, improper line of vision.

The specific gravity is measured by a **urinometer** (Fig. 103), a glass bulb, A, with a weight, B, at the bottom, and a long stem, C, on which is a scale so marked that when the bobbin is dropped into pure water it sinks till the mark 1000 is just at the surface. When put into heavier fluids the bulb will not sink so far, more of the stem will stick out, and on the scale the correct specific gravity of the fluid can easily be read.

One must remember that fluids creep up a little on the sides of the cylinder, *e*, and also on the stem, *d*, forming a

"meniscus." To get the correct reading, therefore, the eye should be on the level of the line, *f*, and look across under the surface of the fluid, thus avoiding both menisci. One looking down from above, as from *g*, cannot read correctly. The cylinder used should be tall and narrow, and stand on a flat table. The bulb, at the time when the reading is made, should not touch the side of the glass. If there is foam on the surface of the urine it is easily removed with a piece of filter paper. Each instrument is accurate at but one temperature, usually "room temperature," about 65° F. On very cold or very warm days the readings will be a little too high or too low. (The specific gravity varies one point for every 3° C. For illustration, if the specific gravity of a urine at a temperature of 20° C. is 1021, that of the same urine at 11° C. would be 1024, and at 26° C., 1019).

In testing specific gravity, a sample of the mixed total twenty-four-hour amount should be used. That is, all the urine passed in the twenty-four hours must be saved, all mixed together, and a sample of this used. The character of the urine varies very much from hour to hour, and very unusual figures may be found if single specimens are tested. Normally, the specific gravity of the total twenty-four hours mixed urine is between 1015 and 1020, but that of one voiding after a heavy meal which contained little fluid may be 1040, and that of a voiding after the ingestion of a large amount of fluid or of some food which acts as a diuretic may be even as low as 1003. If the specific gravity of the total twenty-four hours' urine is about 1040, this would strongly suggest diabetes mellitus; to find it under 1005 would suggest chronic Bright's disease or diabetes insipidus. If the first voiding each morning has a specific gravity of 1010 or less the case usually will be found to be one of chronic interstitial nephritis. A normal person's separate voidings of urine of the day could easily, chosen at different times during the course of the day, give these two readings, but mix up all the urine voided during that whole day and night, and the reading would probably lie between 1015 and 1020.

If we wish to know about **how much solid matter is excreted** in the urine during the day, we multiply the last two figures of the specific gravity by 2.33; this gives the number of grammes of solids in one litre of urine. This, multiplied by the number of litres, gives the weight of solids eliminated that day. Supposing that during the twenty-four hours the person voided 2500 c.c. of urine, and that the specific gravity of a sample of this total mixed urine was 1018. Then that day he eliminated 18 x 2.33 x 2.5, or 104.85 Gm. of solids.

The specific gravity of urine will depend on the **amount of water** and also on the **amount of solids it contains.** One must know both the amount of urine and its specific gravity to judge of the case. Supposing Mr. A. voids 1000 c.c. of urine with a sp. gr. of 1030, and Mr. B. 3000 c.c. with a sp. gr. of 1010. These urines may be quite similar. But suppose Mr. A. voided 1000 c.c. at 1030 and Mr. B. 3000 at 1030. These urines are very different.

In nephritis the specific gravity usually is lower than one would expect, for in these diseases the kidney cannot easily excrete the solids which normally would be eliminated. In **diabetes mellitus,** even while there is polyuria, the specific gravity often runs to from 1025 to 1045 or even to 1060. In **diabetes insipidus** it may run from 1001 to 1005, and yet the amount of urine be very large.

The reaction of the urine when first voided is normally acid. That is, if blue litmus paper is dipped in the urine its color will be changed to red. The reason for this is that we eat much food (meat) whose ashes are acid. The ashes of vegetables are alkalies, and hence herbivorous animals void an alkaline urine.

The reaction of the urine should be tested just as soon as it is passed, for on a warm day it becomes alkaline if allowed to stand for even a few minutes. The multitude of bacteria which at once (unless it had been collected under aseptic conditions) gain access to the urine and rapidly multiply there break up the urea, which itself is neither alkaline nor acid, and set ammonia free. Ammonia is a strong alkali,

which explains its very disagreeable odor. Sometimes even fresh urine is alkaline. This occurs especially when there are growing in the pelvis of the kidney or in the bladder certain bacteria which can break up the urea before the urine is voided. (Some germs, e.g. Bacillus tuberculosis, do not do this and so are found in an acid urine.) Sometimes, immediately after a meal which stimulated the secretion into the stomach of much very acid gastric juice, the urine will, for a few minutes, be alkaline; the reason is the unusual amount of acid taken from the blood by the gastric mucosa.

The **color** of the urine sometimes aids in diagnosis. The coloring matter of normal urine comes ultimately from the blood. The hæmoglobin of worn-out red corpuscles is removed from the blood by the spleen. Its iron is split off and saved; the rest becomes bilirubin, the coloring matter of bile. In the intestine bilirubin is slightly modified to form "hydrobilirubin," and some of this is reabsorbed into the blood and excreted in the urine as "urobilin."

The higher the specific gravity of urine the deeper, as a rule, its color. Diabetes mellitus is an exception, for here the color is pale. In some anæmias the urine is pale because so little hæmoglobin is destroyed, in others it is dark because so much is destroyed. The color depends also on the food we eat and on the medicines we take, and the latter may produce startling colors, such as green, or black, or deep blue. **Bile** in the urine, which happens in most cases of jaundice, is best recognized not by the color of the urine, but by that of its foam when shaken, for even the darkest urines have a white foam, while a yellow foam means, in nearly every case, bile. The presence of a little blood in the urine gives it a smoky appearance; that is, it is turbid and has a blackish-red tint. After carbolic poisoning, and in some cases of constipation the urine is **almost black.**

The **translucence** of the urine is often important. Fresh acid urine is clear. Soon after standing, however, there appears in it a feathery cloud, the "nubecula," composed of the mucus washed by the urine from the mucous membranes

lining the urinary passages. If a person has not been drinking much water, but has been eating considerable meat, and the day is cold, his urine may present a remarkable appearance. A heavy precipitate forms which at first makes it very milky and which soon settles on the bottom of the vessel as a heavy layer of white, pink, or very red sediment, the so-called "brickdust sediment," which at once disappears if this turbid urine is warmed. This sediment sometimes frightens a normal person, particularly as patent-medicine advertisements have given this as a sign of serious diseases. It is however merely the **urate sediment**—that is, a precipitate of the salts of uric acid which are present in every urine, and which are deposited whenever the urine is a little concentrated and the day is cold, and although common in the urine of fever patients, it means nothing at all abnormal in the kidneys.

Only acid urine is clear. When, because of the bacteria, it becomes even slightly alkaline on standing, the phosphates at once separate out forming a white sediment, while the fluid is soon turbid with the enormous number of bacteria growing there. This is the **phosphate sediment,** all of which (but not of course the bacteria) is at once dissolved if enough acetic acid is added to change the reaction of the urine to acid. A similar sediment is present in freshly voided urine from an infected renal pelvis or bladder, should it be alkaline. While in other cases, with the alkalinuria not due to bacteria (see page 280), the phosphate sediment is present in the freshly voided urine; but in these no cloud of bacteria is present. If a urine is clear when voided, no later-appearing sediment has any importance. If a urine freshly voided is not clear, but contains a sediment which will not disappear on warming or on the addition of acetic acid, the sediment does indeed mean something. It means usually either bacteria, blood, pus, or casts, and is called an **organized sediment.**

It is very important for the nurse to know **how to prevent changes in the reaction of urine,** since the examination of a decomposed specimen is difficult and untrustworthy. Each specimen should be preserved in a perfectly clean and tightly

corked bottle. A few crystals of thymol or gum camphor will often keep the urine in good condition, or, better still, a teaspoonful of chloroform, or a few drops of formaline. Which should be used depends on what use the doctor is to make of the urine. If the chemical examination is most important, chloroform is best; if the microscopical, formaline. Whatever preservative is used, the urine should be kept on ice.

It is also of value to know **how to make clear for chemical examination a specimen turbid** with bacteria, since filter paper is of little use. The best method is to add to the urine a small quantity of infusorial earth (called also Kieselguhr), about a tablespoonful to 400 c.c. of urine. The urine is then well stirred up and filtered through ordinary filter paper. The germs are held mechanically by the earth.

Diseases of the Kidneys: Nephritis

Uræmia. Of all the disturbances of the kidneys uræmia is one of the most dreaded. It is the highest expression of renal insufficiency and therefore develops in connection with, or as a result of, kidney disease. It may come on suddenly with coma, convulsions, and death, or more slowly with, at first, nervous or mental symptoms.

We know that a uræmic patient is being poisoned; the symptoms prove that; but what that poison is, no one yet knows. While it may possibly be some substance which the kidneys should, but do not, remove, yet, if it is, it quite certainly is no one of the products of normal metabolism which we know, although in most (but not all) coma cases we do find these products (total nonproteid nitrogen, including urea, uric acid, and creatinin) greatly increased in the blood. There would seem to be at least two of these hypothetical toxins, the one which causes coma and another which causes convulsions. Formerly it was thought that the accumulation of urea (hence the name) in the blood was to blame, and this explanation is natural, since urea is the most abundant and best known constituent of urine; some think that the liver has an important share in the production of uræmia; others, that the reason for the mental symptoms, coma, and convul-

sions is to be found in the brain (œdema etc.); while in many cases terminal systemic infections might explain the picture. How important in the causation of uræmia the kidneys themselves are, it is hard to say. Certainly we cannot, from the examinations of the kidneys after death, tell whether or not the patient had died in uræmia. Sometimes the kidneys of uræmic cases show much less disease than those of other cases of Bright's disease who died without uræmic symptoms.

Uræmia is the name not of one single condition but of several. The removal even of all the kidney tissue a healthy person has (which too often has happened, a fused kidney being mistaken at operation for a tumor) does not produce a typical uræmia, although it does produce the most perfect anuria possible (the most complete renal insufficiency), and therefore would give the best possible chance for the poisoning of the body by the constituents of normal urine. These unfortunate persons have, for even as long as two weeks, no symptoms at all except weakness. Then begins a rapidly developing coma, often without convulsions, and, in a few hours, death. Typical cases of uræmia usually have some evidences of cerebral trouble (dyspnœa, Cheyne-Stokes respiration, retinal œdema, and a spinal fluid under high pressure, the removal of which gives relief) which some ascribe to œdema. In still a third group of uræmic cases with marked cerebral features, the toxic and convulsive features are early and spectacular:—flashes of light (hence the name "eclampsia"), blindness as an early feature, hallucinations, delusions, and mania. This form resembles the eclampsia of pregnancy, a different although related condition.

It is the **accumulation in the blood of the normal urinary constituents** which gives us our best warnings of developing renal insufficiency. A patient is in imminent danger of uræmia in whose blood the creatinin has reached 5 mg., the uric 5 mg., the urea 80 mg., and the total non-proteid nitrogen, 120 mg. per 100 c.c.s. of blood.

Another warning of uræmia is given by certain **renal functional tests**, especially that which uses phenolsulphone-

phthalein. Half an hour before this test is made the patient is required to drink from 300 to 400 c.c.s. of water in order to be sure of free urinary secretion (failure to do this has given some very deceptively low figures). Just before the injection, he is asked to empty his bladder. Six mg. of the above mentioned dye are then injected subcutaneously or intravenously. The urine is then collected exactly at the end of the first, and again exactly at the end of the second, hour after the injection. Any other voidings during these two hours are added to the proper specimens. If there is any doubt as to the patient's ability completely to empty his bladder he should be catheterized. All the urine secreted during the first hour is poured into a one-litre measuring flask, an alkali (25 per cent NaOH) is added in sufficient amount to turn the dye present in the urine a brilliant red. It is then diluted exactly to 1000 c.c.s., thoroughly mixed, and its color measured with a colorimeter. The urine secreted during the second hour is treated in exactly the same way. We thus may determine the exact amount of the dye eliminated during the first hour and during the second hour after the injection. A normal person will eliminate from 50 to 60 per cent of the dye during the first hour and from 20 to 25 per cent during the second hour; that is, from 60 to 80 per cent during the first two hours. Often before uræmia the two-hours' output of this dye sinks to 10 per cent or even to 0.

Every nurse should know the **early signs of uræmia.** The great majority of cases begin with one or more of a group of symptoms—headaches, drowsiness, vomiting, restlessness, mental wandering, a foul breath, etc. If we then begin active treatment, the uræmia may disappear. Sometimes the onset is sudden. A person who apparently has enjoyed excellent health, who may never even have suspected that he has kidney trouble, suddenly falls in a convulsion, or is found unconscious, or becomes acutely insane with dangerous delusions. The majority of cases of uræmia, however, develop during the course of a well-recognized chronic nephritis. These patients gradually become drowsy and complain of

headache. They may vomit. Their respiration becomes Cheyne-Stokes in character, and an ever-deepening coma follows, sometimes with convulsions, which may be mere muscle twitchings, or severe spasms often quite similar to those of epilepsy. Unless treatment is successful death soon follows.

The treatment of uræmia should be prompt and vigorous. Early active purgation is imperative, using salines, or, if coma has already begun, stronger purges, even croton oil; and warm baths, even one or two sweat baths a day. The reason why these so often help is, some say, because they make the skin and bowels do some of the kidneys' work; but others believe, because they bring the blood to the surface of the body, therefore away from the brain and the kidneys. We know that venesection (removing usually between 400 and 500 c.c.) may help these patients, perhaps because by so doing we remove some of the poison which is in the blood, but perhaps because it brings about a consequent redistribution of the blood. By the administration of salt solution (containing also 1 per cent sodium bicarbonate and 2 to 4 per cent of glucose) either into the rectum, using the slow drip method, or intravenously, we try to dilute what poison remains. Whatever the explanation, there is no doubt that purging, sweating, and bleeding all help considerably. For the restlessness and dyspnœa often present one gives chloral or, if necessary, morphia. Lumbar puncture, and removing, if it is under pressure, considerable of the spinal fluid, will give temporary relief. As the result of such treatment, the patient may regain consciousness and the immediate danger is over. The further treatment is that of any case of severe Bright's disease.

Nephritis is a term which might be expected to designate inflammation of the kidneys. But this would be a very inadequate description of the diseases called nephritis. True, the kidney shows changes some of which are definitely inflammatory in nature, but also other changes which it is hard to explain in this way, including degenerations, fibrous tissue formation, etc. Again, in "nephritis" the lesions are

by no means limited to the kidneys, but early (and some say earlier) involve other organs also, such as the blood vessels, the liver, the brain, etc. Certainly in nephritis the body as a whole is affected.

In acute nephritis the kidneys are large, swollen, fatty and congested. An exudate of blood-plasma, red corpuscles, and leucocytes escapes from the capillaries and infiltrates the kidney substance. The renal cells are so injured that they cannot do their work properly; many die.

An acute nephritis is probably always due to some poison which directly injures the renal tissue. This poison may be a drug taken in too large amounts, as mercuric bichloride, turpentine, potassium chlorate, or carbolic acid. Such chemical poisons are eliminated by the kidneys, which are injured but not necessarily inflamed in the discharge of their duty. Or, the poison (and this is the most common case) may be that of pathogenic germs. Sometimes these germs are localized in organs distant from the kidneys, as in the lung, and the toxins which they produce are carried by the blood to the kidneys. In other cases, these germs settle in the kidneys themselves, and there liberate their poisons. This explains the nephritis which develops as a complication of scarlet fever, smallpox, acute tonsillitis, typhoid fever, diphtheria, septicæmia, and abscess. In other cases the poisons which injure the kidney are the products of diseased tissues. The nephritis which follows bad burns and that which accompanies some extensive skin diseases may have some such origin, although it is possible that here also germs are in part responsible. Perhaps the commonest cause of an acute nephritis which is not a complication of some other disease is exposure to cold or wet. The explanation of this we do not know. Either ordinary acute tonsillitis, or "a simple sore throat," or buried infected tonsils which for years have given no symptoms, are now considered the starting-point of very many cases of acute nephritis. These tonsils seem to be the "portal of entry" through which many germs enter our blood.

A truly acute nephritis seems to be almost exclusively a

disease of childhood. In favor of this idea is the fact that most of the cases of adults diagnosed as acute prove later to be merely acute exacerbations of a latent chronic nephritis which had begun in youth. This point is very important, since, if proved to be true, we should raise the slogan "protect the kidneys of children and so save those of the adults."

The symptoms of acute nephritis are very variable. Sometimes the disease begins suddenly, and even violently, with fever, occasionally accompanied by a chill, headache, dizziness, nausea and vomiting, pains in the back and, within twenty-four hours, swelling of the face and feet, and such weakness that the patient cannot walk. It is seldom, however, that the symptoms of onset are as definite as this, and acute nephritis is a good illustration of those dangerous diseases which sometimes progress to serious grades before the person suspects that he is sick. Too often an uræmic convulsion, or sudden death, or, after months or years of apparently good health, the evidences of chronic nephritis, reveal the presence of a disease which has been developing for years. In the majority of cases, however, the patient, from the first, notices that both ankles swell a little, that his face is pale, that his eyelids are a little puffy, but otherwise he may feel so well that he argues himself into the belief that these are unimportant. Some patients are blind for a while, yet without any evident lesions in the eyes; others develop a retinal hæmorrhage or an albuminuric retinitis. The blood pressure usually is a little raised. If it is much increased, however, (i.e., over 180 mm. Hg.), one should suspect that the condition is an acute exacerbation of a latent chronic nephritis. The phenolsulphonephthalein test is of value in these cases, since, early in a case of acute nephritis, the elimination of the dye is reduced to from 30 to 20 per cent or lower, and in another two days may be higher than normal. Any increase in the blood plasma of the total nonproteid nitrogen will be slight unless the case is severe.

The urine output will be scanty; there even may be none (anuria) for a day or two. Usually, however, the patient,

early in the disease, passes from 50 to 200 c.c. daily of a highly colored, cloudy urine with a specific gravity often between 1020 and 1025 (i.e., low considering the total amount) and with a thick sediment of red blood cells, leucocytes, and all kinds of casts, but especially of the epithelial, blood, and coarsely granular varieties. This urine contains large amounts of albumin. As the patient improves, the amount of urine increases while that of the albumin and of the sediment diminish. Sometimes, perhaps oftener than we think, the patient gets entirely well, provided we remove the focal infection (e.g., the tonsils) which, if retained, might keep the trouble alive for years. If, on the other hand, the condition gets worse, the anæmia and dropsy increase, and, finally, uræmia develops. In still other cases, the patient seems to get well, but the disease develops insidiously into a chronic nephritis.

The nursing care and treatment of nephritis deserves careful study since of medical or surgical care there is little and practically all of the active therapy we have is in the field of the nurse. The treatment must be prompt and vigorous, even though the patient says he feels well. First, **the tonsils if infected should be removed and all other easily operated focuses of infection should be drained.** This can be done safely, even while the condition is acute, if the operations are rapidly and skillfully performed. At least, to operate is often the lesser of two evils. The care of mouth infections is especially important.

While the nephritis is acute, as shown by the slight fever and the presence of the blood in the urine, rest in bed is essential. This includes not only physical rest but the elimination of all worry and all causes of discomfort and restlessness. **The horizontal posture is very desirable,** as is evident from the fact that in all conditions with albuminuria the amount of albumin eliminated is always greatest when the body is erect or semi-erect and is less or even absent when the body is horizontal. This would seem to depend on the circulation of the kidneys and manifests itself after

even a few minutes' rest in bed. Too long rest in bed, however, is itself not without harm, and after the more acute stage is over the lesser of two evils may be to allow some activity, for there is much truth in the general criticism that in the past our treatment may have saved the patients' kidneys but sacrificed his life.

Since the function of the kidneys is to excrete the ashes of food, **the diet in nephritis** is particularly important and should be of those foods producing ashes which are the least possible irritating to the kidneys.

Rest for the kidneys, in the sense in which we use this term of our muscles, **is impossible.** We may lighten their load, but they work on almost continuously, for so far as the kidneys are concerned a man cannot starve. He may swallow no food but he continues to consume his own tissues, so that to the kidneys there can be very little difference between the periods of limited diet and those of starvation. Nevertheless, by carefully regulating the diet and avoiding all foods whose ashes present a renal problem we can lighten their work considerably. Proteins are restricted to an amount just sufficient to meet the body needs. Reduce the intake still more and the body will make up the deficit from its own tissues. On the whole, the proteid of **milk would seem the best,** perhaps, partly, because it is purin-free, provided this be given in sufficient quantities. The milk may be sweet, or it may be buttermilk, which for some is much better. As a rule it is best drunk hot. **Eggs** also are a purin-free source of protein (the former prohibition of eggs, especially raw eggs, was based on a misconception of biochemistry which identified egg albumin and serum albumin), while the rest may well be gained from fruits and bread. **Certainly all purin-rich foods should be avoided,** as kidney, liver, sweetbreads, brain, and an excess of meat, and of the meats allowed those of the young animal and young fowl have the advantage of being relatively freer from extractives than are those from older animals and birds.

Again, **many vegetables contain mineral salts** which are

hard on the kidneys. **Beef extracts and meat soups** should be avoided. Beef extract is made by soaking out of muscle all that can be extracted by water, and such extractives are the ashes which that muscle recently produced and which, had the animal lived, would soon have been removed from the muscle by the blood and from the blood by the kidneys. They have little or no nutritive value and, while good stimulants, should be avoided in nephritis. Salt also should be restricted, especially if œdema is present, but **a salt-free diet is injurious** both in health as well as in disease. At the onset of the trouble a **pure milk diet is best.** At least three quarts a day should be given, and better more. Milk is low in sodium chloride, and contains sugar, fat (cream may be added), and proteins the ashes of which are the blandest of all animal foods. It is a great and yet very common mistake to think that one or two quarts of milk a day is a milk diet. Since this alone would not cover the body needs, the patient would fall back on his tissue protein and fat, and the result would be a fat-rich diet with relatively too little carbohydrates. Fruits contain very little mineral salts and so are freely given. Also, they contain fruit acids which in metabolism become alkaline carbonates, which are of definite value to the body. Sugar and fats leave little waste for the kidney to eliminate. If, however, the diet has a deficit of carbohydrates, then the ketonuria which results will more than offset any virtue in the diet. Foods with ingredients which irritate, such as alcohol, celery, onions, radishes, garlic, pepper, mustard, spice and all other condiments are to be rigidly avoided.

Water is also given by rectum, by means of colon irrigations or the Murphy drip. Earlier, when the patient is œdematous, or the urine output too low, fluids are restricted; but for the adult to not less than 1500 c.c.s. per day. **Thirst** may be relieved by ice or water in small amounts. A craving for food, for salt, or for water, which if unsatisfied is always apt to make the patient unhappy, irritable, and depressed, should

be considered as a helpful warning of a tissue need, and in as far as is reasonable, should be satisfied.

The condition of the skin is of vital importance. While the old idea that the skin through sweating can act vicariously for the kidneys is hardly correct, yet these two tissues are definitely related, and to treat one is to treat the other also. Unfortunately, the amount of solids eliminated through the skin (and always proportional to the amount in the plasma) is almost negligible for therapy. Nevertheless, to keep the skin warm and moist will definitely improve the circulation of the kidneys. Rest in bed between blankets, daily cleansing baths, and massage aid the kidneys greatly. All exposure to cold air and cold water is to be avoided, since this increases the congestion of the kidneys, contracts the blood vessels of the skin, and raises the blood-pressure. Fresh air without exposure is desirable. The care of the skin is important, also, because of the danger of bedsores, since the patient is long confined to his bed, is obliged often to sit up constantly in one position, while the œdema and general cachexia of the skin and subcutaneous tissue favor their production.

The elimination by the kidneys may be stimulated **by keeping the patient in a horizontal position,** by forcing fluids by mouth or by rectum, and by the local applications to the lumbar regions of counter-irritants, stupes, mustard plasters, dry cups, etc., which relieve inflammation, congestion and pain. **Hot colon irrigations** not only supply fluids but have a beneficial effect on the kidneys. **Alkaline diuretics,** such as sodium citrate and potassium acetate, are of rather doubtful value, while diuretics which directly stimulate the renal cortex do more harm than good.

While rest for the kidneys **by elimination through all other channels** is theoretically desirable, yet this is possible only to a limited degree; nevertheless, all of the measures employed in the past to accomplish this are excellent treatments since they tend to stimulate the circulation and to improve the functions of the skin. Hot fluids by mouth, hot baths,

hot packs, and diaphoretic drugs, if not carried too far, all benefit the patient, because of their effect on the general and therefore renal circulation; nevertheless, the actual elimination of metabolites through the skin is almost negligible. Profuse sweating, on the other hand, may dangerously concentrate the uræmia-producing substances in the blood, and too great heat may have a depressing effect on the heart and nervous system.

The bowels are kept loose by the use of those cathartics which cause watery movements, especially the salines, jalap, etc.

Headache is relieved partly by increasing the elimination, partly by the local applications of heat or cold to the head, sometimes by lumbar punctures, and usually by the administration of such drugs as phenacetin and antipyrin.

Backache may be relieved by the local applications of heat, and by massage. **Dyspnœa,** if by cerebral origin, can be relieved by morphia or related drugs. That due to fluid in the pericardial, pleural, or abdominal cavities, must be relieved by its removal by tapping of these fluids. The nursing care of orthopnœic cases is extremely important as well as difficult, because of the clumsiness of the œdematous limbs, the insomnia, and the general physical and mental discomfort of the patient.

Œdema is relieved by any measures which improve the circulation, by free purgation, and, if extreme, by aspiration needles in the skin (page 298). Little is gained by rigidly restricting salts and fluids.

When the kidneys shall have recovered their ability to eliminate within normal time the water, as determined by the tests for retention of water (the daily output of urine should be at least seventy per cent of the total intake of fluid and the specific gravity of the earliest morning specimen should be above 1010) and the disappearance of œdema, **fluids are usually forced** in order to dilute the waste products, to flush them from the system, and, by diluting the urine, to lessen the irritation of the kidneys. Water, lemonade,

orangeade, and imperial drink are given by mouth. The diet also should be increased by the addition of a little bread and butter, custards, a soft boiled egg, and, later, by even a little meat once a day. The patient is never allowed the viscera (kidney, liver, sweetbreads, or brain), nor is he given pepper, mustard, spice, or alcohol. Iron should be given for the anæmia (best as Basham's mixture). **After the albumin has been absent** for a few weeks the patient may sit up, and later may exercise more and more. In many cases, however, unless the cause (tonsils, sinus infection, alveolar abscesses of teeth, infected mediastinal lymph nodes, etc.) has been entirely removed, the albumin will not entirely disappear and the patient must plan for years of treatment for a subacute or chronic nephritis.

In acute nephritis all the tissues of the kidney are affected, but in differing degrees. In adults a classification based on these differences is difficult, but in children we may separate at least these fairly well marked, and yet not perfectly distinct, types: tubular nephritis, glomerular nephritis, and intestinal nephritis. One recognizes the **tubular form** by the sudden appearance of **œdema of the face, feet, and then of the whole body,** which soon becomes **marked,** more so than in other conditions. This may come on without preceding symptoms or shortly after an infection of the upper respiratory tract, and sometimes also with nausea and vomiting. The whole body may be bloated with dropsy, the eyes closed, and the abdomen and pleural sacs filled with fluid. Yet the patient feels well, not nearly as sick as he looks or as he is. This dropsy fluctuates markedly from day to day, and without apparent cause, and may entirely disappear and then later recur. The attacks of tubular nephritis last from two months to three years, an average of eight months. The amount of urine voided varies greatly, according to the progress of the œdema, but in general is almost normal. But the amount of albumin it contains may be surprisingly great, almost the greatest ever found. So much may be present that the urine in the test tube becomes solid when boiled.

Nevertheless the renal functional test is usually normal (average 57 per cent in two hours) and sometimes even excessive (95 per cent). Casts in the urine are numerous, but, and this is important in diagnosis, **practically no red blood cells** can be found. Blood chemistry shows that the plasma has been depleted of albumin but that there is no accumulation in it of urea, uric acid, creatinin, etc. There is also no rise of blood pressure.

Some attribute this form of the nephritis of children chiefly to infection of the nasal sinuses, but in most cases no cause can be discovered.

So slight, in tubular nephritis, are the evidences of acute inflammation that the term **nephrosis** has been proposed to signify that the trouble is more a degenerative process than an inflammation. This form of nephritis is relatively rare.

The treatment of tubular nephritis is rest in bed and a light general diet during the periods of œdema, but more activity and a freer diet when the œdema is less intense (even though the albuminuria remains marked). There is little gain from the over abundant albumin diet proposed by some. An excessive ascites or hydrothorax should be relieved by tapping. Any infection, as of the sinuses, should receive prompt attention. Diuretics would seem to control the œdema best; thyroid extract has some value; purges are important; calcium chloride, etc., may be tried. Many of these patients get well.

Glomerular nephritis is a much more serious condition and causes greater prostration than does tubular nephritis. It is marked also by much **less œdema** (sometimes none) and always, and this is the distinctive point of diagnosis, by the presence of considerable blood in the urine. In children another group is recognized, **interstitial nephritis,** in which there is no œdema of the skin and no blood in the urine, only albuminuria as a conspicuous sign. The treatment of glomerular nephritis is that of acute nephritis (page 298). It certainly should be much more rigid than that of the tubular variety, with long rest in bed, milk diet, free purga-

tion and general supportive treatment. That of the **interstitial variety** is chiefly symptomatic.

Tubular nephritis in young adults is represented usually by the so-called **chronic parenchymatous nephritis.** That is one of the most severe forms of Bright's disease. It attacks young adults especially. While it certainly is an acute condition, yet it begins so silently, it lasts so long, and its acute symptoms are of so mild a grade, that subacute is the better term. It formerly was, and still is, called parenchymatous nephritis, but this is an unfortunate term. Formerly one tried to differentiate sharply between diseases of the parenchyma of an organ (in this case the kidney cells) and those of its interstitial tissue (the supporting structures of connective tissue, the blood vessels, etc.). No such sharp distinction, however, can be made, and the chances are that the so-called interstitial changes are merely the evidences of healing following the destruction of the epithelial tissues. Some classify the subacute cases in young adults as we have the subacute nephropathies of children, but in young adults the distinctions between the tubular, glomerular and interstitial varieties are not so definite.

The kidneys of these patients are larger than normal—even three times the normal size—and the cortex, instead of being about 6 mm. in thickness, is, in extreme cases, even 12 mm. The renal cells are swollen and filled with granular debris and fat. The pyramids are much congested. Here and there throughout the kidney, small hæmorrhages are always found. There is considerable increase in the renal connective tissue.

The urine is at first a little decreased in amount, or it may be normal in quantity, but, as the case improves, it usually increases even to from 6 to 8 litres a day. It has a dirty yellow color, is very turbid even when fresh, since it contains very many casts of all kinds, especially of the epithelial, granular, fatty, and waxy varieties, many renal epithelial cells, pus-cells, and always some red blood corpuscles, all of which settle as a thick sediment. Large

amounts of albumin are present. The normal constituents of the urine are diminished since the diseased kidneys cannot properly perform their function. In the majority of these cases the phenolsulphonephthalein test shows an elimination of but 30 or even 10 per cent of the dye in two hours, but among these cases is one group, (perhaps an early stage of all) with so much hyperpermeability that from 80 to 95 per cent of the dye is eliminated in two hours. The amount of non-proteid nitrogenous substances in the blood is usually normal and increases only in proportion as the patient verges on the border of uræmia.

When very ill, these patients present a striking picture. **The skin has a very pale, pasty color;** the whole body sometimes, and always the face, lower extremities, and dependent parts of the body, **are swollen with dropsy.** Often the finger can be pushed fully a quarter of an inch into the water-logged skin of the legs. **Water collects** in the abdominal cavity (ascites) which greatly distends the abdomen. It collects also in one or both pleural cavities (hydrothorax, water on the chest) and the patient is short of breath and must sit upright (orthopnœa). Water may collect in the pericardial sac (pericarditis with effusion) and the patient is in consequence very short of breath, cyanotic, and has a weak pulse, especially during inspiration. During inspiration the pulse normally is strong and regular in rate but a little faster than during expiration (the respiratory variation, or sinus arrhythmia). One test of pericarditis with effusion is that during inspiration the pulse waves are weak and the rate slowed because of the loss of one or two of the beats (pulsus paradoxus, since contrary to normal).

The cause of this dropsy is not known. One theory ascribes it to the retention of salt, and therefore suggests a salt-free diet. But this theory has very little to support it, and a truly salt-free diet would be dangerous. (By this we mean a diet with all of the salt possible washed out of the meat and purposely omitted from the bread, etc.). Salt-poor diets, however, are to be recommended for the well as

well as for the sick, since many well persons use nearly twice as much of this condiment as they need. Others say that in nephritis the reaction of the tissues changes, and that as a result the cells swell with water. But this would be only a partial explanation. Another reason suggested, and with some shadow of truth, is that the heart in nephritis is never at its best, and that the resulting poor circulation may explain the accumulation of fluids in the body cavities. All of these reasons may be true, and yet all added together are an inadequate explanation.

The blood pressure in this form of nephritis often is high, the vessel walls sooner or later show considerable arteriosclerosis, as a result of which hypertrophy of the heart soon begins. The patient often has severe diarrhœa and may vomit frequently. He always is toxic and yet seldom is uræmic until just before death.

The causes of this form of nephritis are many. Sometimes it begins as an acute nephritis which did not respond to treatment, but in the majority of cases such an acute trouble, if present, certainly gave no symptoms. Often we get the history that the patient has had scarlet fever, tonsillitis, acute sinusitis, malaria, or any one of many acute fevers, during which illness the ankles were a little swollen, and the convalescence from which was slow, and that his urine, after this illness, always has contained a trace of albumin. That is, this patient, ever since that fever, has had a latent, insidious case of Bright's disease. Of course some cases of acute nephritis will, in spite of the best of treatment, develop into this form of the disease, but careful treatment will greatly reduce this danger. Other cases of subacute parenchymatous nephritis are due to lues; others, and the largest kidneys of all belong in this group, are due to longstanding tuberculosis, although these huge kidneys themselves are not tuberculous; others, and some say this is the most important cause of subacute parenchymatous nephritis, are due to the habitual use of alcohol, even in small amounts.

The prognosis of this disease is bad. A few cases will

improve, their nephritis assumes the chronic form next to be described, and they may enjoy fair health for many years. The great majority, however, slowly fail and die in from one to two years, often in a uræmic condition.

The treatment of parenchymatous nephritis is exceedingly important, and the nurse's share especially so. Any infection of the tonsils and nose, and especially lues, should receive proper treatment. The patient is kept in bed, propped up, and made as comfortable as possible. The diet consists chiefly, but not entirely, of milk. Some variety of foods, however, is desirable, as a long and limited milk diet will in time add its injury to the condition. The patient should always receive sufficient proteid and, some say, an excess if the case suggests the nephrosis of Epstein (page 294). He should be encouraged to drink as much fluid as he can eliminate. Warm packs to the point of sweating, but not longer, should be given daily. When the œdema is severe the skin of the lower extremities is pierced with hollow needles to which are attached rubber tubes which lead to a bottle under the bed. These needles may remain in place even for days. The head of the bed is kept elevated, and, surprisingly enough, much of the fluid causing the œdema even of the face will drain downwards and out into the bottle. The nurse must watch those tubes with particular care since infection around them would be very easy and serious. The collections of fluid in the body sacs, whenever the patient is much distended or is very short of breath, must be drawn off by tapping. Marked shortness of breath, delirium, cyanosis, and a pulse which during inspiration weakens and even loses a beat, suggests that the pericardium should be tapped at once. The nurse should watch for these symptoms, as well as for those which suggest uræmia (page 284).

Of drugs, caffeine, diuretin, or theocin are often given as diuretics (although many think they do more harm than good) and digitalis, to aid the heart. The bowels must be kept freely purged, using Epsom salts, or jalap. The patient should always receive iron, best as Basham's mixture. What-

ever medicines will add to the patient's comfort are justified, for the course is long, the condition serious, and the prospect bad. If uræmia develops, this requires its own special treatment (page 285). Should the patient improve, the treatment should relax very gradually.

Chronic nephritis with œdema is a diagnosis which covers a large group of cases with nephritis which last for years

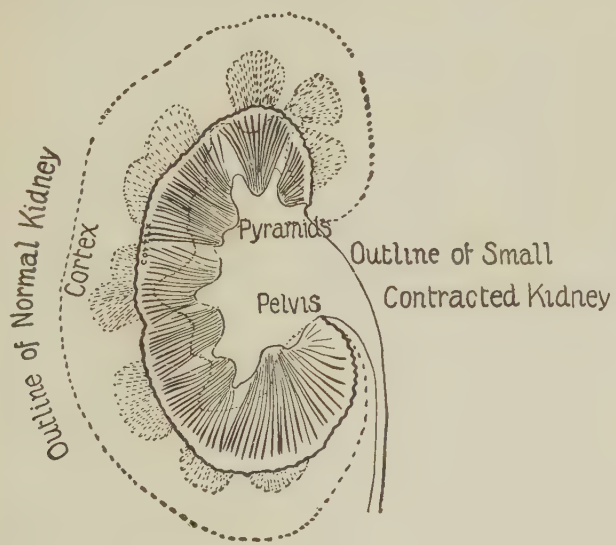


FIG. 104.—Chronic interstitial nephritis.

and which have very few clinical symptoms other than œdema. The kidneys of these cases present all the transitional stages between subacute parenchymatous and chronic interstitial nephritis. Many of these patients are able to lead active lives and would scarcely be aware of their condition were it not for the swelling of the lower lids and of the feet, worst at the end of the day, and the frequent micturition, especially during the night. They are anæmic. They have frequent gastrointestinal disturbances. Their blood pressure may be high or normal. Why high, the reason is not known. They usually are short of breath and occasionally

dyspnœic. Some have a sudden temporary dimness of vision due to a retinal hœmorrhage. These cases suffer especially from the consequence of their general arteriosclerosis, always marked, and from their cardiac symptoms, especially if their arterial pressure is high.

The urine always contains albumin, usually more than traces, and casts, especially of the hyalines and finely granular varieties, and a few red blood cells. The renal functional test shows a more or less reduced elimination of the phenolsulphonephthalein, and the nonproteid nitrogenous substances in the blood tend to increase. These cases often have acute exacerbations of their conditions which resemble attacks of acute nephritis. Their treatment will be similar to that of the next variety to be described.

Chronic interstitial nephritis, cirrhosis of the kidney, and chronic nephritis without œdema, are names given to a very chronic form of nephritis in which much of the renal tissue is gone and its place taken by scar tissue (Fig. 104). There is usually in progress also a slight acute nephritis (although not enough to cause symptoms) and it is this which explains the disease, since each of its numerous flareups destroys more and more of the remaining kidney tissue. This is a condition of middle or elderly life.

In a few cases chronic interstitial nephritis can be traced directly back to a subacute nephritis, but this is rare, and few give any history which suggests an acute or subacute renal trouble. More of the cases seem to be the result of general arteriosclerosis, which certainly aids greatly in the development of the condition by limiting the nutrition of the renal cells; yet, as a rule, both diseases would seem to be due to one and the same causes, although each aids in the progress of the other. Long continued infections of the nose, tonsils, teeth, the wall of a chronically infected bowel (seen in cases of chronic constipation), etc., may be important causes. The renal cells certainly are exceedingly sensitive, and when poisoned or starved they degenerate and die, and scar tissue, of no use whatever to the body, takes their place.

The kidneys of some persons are from birth (and sometimes as an inheritance) their "weak organ" and in the strain of life these give out first. Overwork, both muscular and mental, and long continued worry with the resulting high blood tension, injure even normal kidneys, and certainly aid in the progress of a developing renal disease. Some persons, especially if they are gouty, hurt their kidneys by habitual overeating, or by alcohol, or with poisons, as lead, used in their trades.

As a result of this disease the **kidneys are reduced even to about one fifth their normal size.** Their cortex shrinks to a layer one or two millimeters in thickness, and in some places is entirely gone. The surface of the kidney is rough, for the renal tissue disappears in irregular patches, and bands of scar tissue, by contracting, distort the remaining cortex. The glomeruli especially suffer. Many of them have disappeared, and their convoluted tubes as well. One wonders that the renal tissue which is left can function as well as it does. The branches of the renal artery are thickened.

The symptoms of chronic interstitial nephritis are very variable. Some patients with very severe grades of this disease have had for a long time no symptoms at all. They may discover their condition as the result of an application for life insurance. Often it is suggested during a routine eye examination. The first intimation others have is a sudden uræmic convulsion. Many patients merely notice that their feet are slightly swollen at night, but never much unless an acute exacerbation of the nephritis is in progress. Or, the patient may have had many and severe symptoms, but not of renal trouble; rather, symptoms which suggest heart or blood-vessel disease.

Examine one of these patients carefully, and it is likely that **the heart** will be found considerably **hypertrophied, the arteries** very **sclerotic, the blood pressure high,** and the pulse hard because of the arteriosclerosis. He does not "feel well"; he is losing weight and strength; he suffers from shortness of breath, terrible headaches, fearful dyspnœa at night, and

disturbed eyesight, due to the occasional retinal hæmorrhage; later, the symptoms of uræmia may appear.

Why, in nephritis, especially in the chronic types but also in the acute, **should the blood pressure be so high,** even twice the normal, itself causing more and more arteriosclerosis and still more renal trouble, is not known. One theory is that certain ashes of food which the diseased kidneys fail to remove properly from the blood stimulate the small arteries all over the body to contract. This would enormously increase the work of the heart since it has to pump against this increased peripheral resistance. Another idea is that there are but few glomeruli left, and that the heart is stimulated to pump as much blood as possible through these few. These two ideas both may be true. A third idea is that the cause of the arterial hypertension is not in the kidneys but in other organs also involved in this disease, especially the brain.

Among the common symptoms of chronic nephritis are **polyuria, frequent micturition especially at night,** and a **low specific gravity of the morning urine.** One reason for these last two symptoms is what is called "retention of water with nocturnal elimination." Normally, we void the most of the water within two or perhaps three hours after we drink it. In these cases, however, the water, because of the disease, cannot all be eliminated during the day and so is retained until the body is at rest. Finally, these cases show fixation of the specific gravity of the urine. That is, their urine does not show the marked variabilities of concentration due to what we eat, drink, or do, but is all of practically the same composition, regardless of the patients' activities. **The renal functional test** shows a tendency to **a low elimination of the dye** and with it a **marked accumulation of nitrogenous bodies** in the blood. The elimination may finally sink to 0 in two hours.

The urine will be increased in amount, usually to 3,000 to 4,000 c.c., but sometimes even 12,000 c.c. a day. It is pale in color, clear, and **of low specific gravity**—from 1005 to 1012,

especially in the morning. This is very important, for, if the urine voided soon after waking has a specific gravity of 1010 or less, one may safely make a diagnosis of this condition even in the absence of albumin. It never contains more than **a trace of albumin,** except during the occasional acute exacerbations, and is sometimes absent for weeks, or is present only in the afternoon; and the urine contains so few casts that they are difficult to find. It is in this group of cases that uræmia is most likely to develop.

The treatment is simple: to avoid aggravating the condition and to spare the kidneys as much work as possible. This condition is apt to last from 10 to 15 years, and the patient naturally will insist on remaining active. Any cause of acute exacerbations should if possible be removed. The patient should eat lightly, avoiding all the foods mentioned on page 289, should use meat but once a day, and yet not starve himself. He should drink no alcohol at all. He must refrain from all severe work, physical or mental. Colds must be avoided and any infection of the nose, throat or mouth treated carefully. Climate is most important. A warm, agreeable one may prolong life considerably. Hydrotherapy (warm baths and electric light baths) give great comfort. The skin and bowels deserve special attention because these tissues seem to have some definite relationship to the renal epithelium. The heart condition must be watched and treated. The rule of life for this patient is "temperance in all things."

Other Diseases of the Kidneys

Floating kidney was formerly one of the bugbears of the popular mind, and to it have been attributed all kinds of pain. The lower half of the kidneys of thin women, and especially of the right kidney, is easily felt during inspiration, since normally the kidneys descend with each deep breath, and sometimes considerably so. Such is called a **palpable kidney,** and is quite normal. One cannot, however, no matter how deeply the patient breathes, reach with his fingers above the upper pole of a palpable kidney and so hold it down during expiration. When this is possible the

condition is one of **movable kidney**, a not quite normal condition. The normal kidneys are held in place by their fat capsules, and for this reason persons who have suddenly lost considerable weight often have a movable kidney. The relaxation of the abdominal walls which mothers suffer is another cause. The right kidney is more often movable than the left. A movable kidney when released during expiration snaps back into its normal position. One which does not do so but which tends to remain in whatever position one pushes it is called a **floating kidney.**

Palpable kidneys give no symptoms at all. There may be some symptoms with (but not often due to) a movable kidney, and none from a floating one; but let a nervous woman suspect she has one of these conditions and plenty of distressing symptoms will soon appear, dragging pains in the side, dyspepsia, and a whole gamut of nervous disorders.

Dietl's crises, which seldom are met with, are especially interesting, since they do seem to be due to a movable kidney, that is, to one which rotates on its axis enough to twist its ureter and blood vessels. The results are paroxysms of very severe pain with nausea, vomiting, chills, fever, and collapse.

The treatment of a mild case of movable or floating kidney (and most cases are mild after the patient is somewhat reassured) is to increase the patient's weight, for a few extra pounds of fat in the abdomen are better support for a movable kidney than is silver wire. If necessary, a well-fitting binder with a pad can be adjusted to hold the kidney in place. The nervous condition, especially, of these patients must be treated. If very severe, as when a twist of the kidney frequently closes temporarily its ureter and pinches its blood vessels, an operation may be justifiable, but probably will not be as successful as a well-conducted "neurasthenic-cure" would be.

The pelvis of the kidney (including the calyces) is the wide sac into which the urine from the pyramids is poured, and which, by narrowing down to a small tube, becomes the

ureter. The pelvis has very thin walls whose inner surface is lined with the same kind of mucous membrane as lines the ureter and bladder.

The pelvis of the kidney and its calyces (Fig. 105) will be distended by the partly dammed back urine (combined with the secretion of its mucous membrane) when the ureter is somewhat obstructed. If, in such a case, no inflammation is present, the fluid is clear and the condition is called

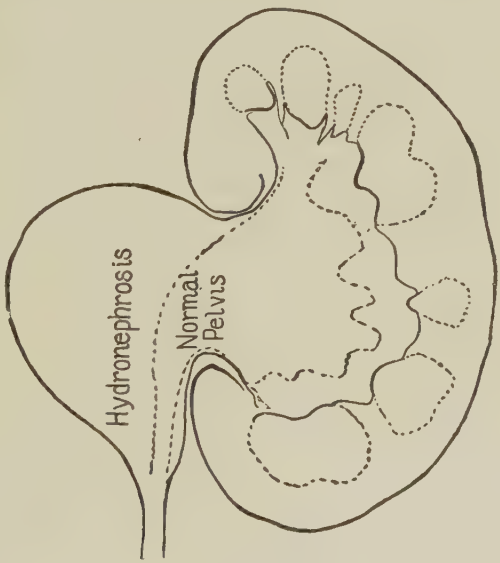


FIG. 105.—Hydronephrosis.

hydronephrosis. If, later, this pelvis becomes infected, its contents, instead of a clear fluid, will be pus, and the condition is then called **pyonephrosis.** To cause any dilatation of the pelvis of the kidney, the obstruction of urine must be gradual, partial, or intermittent. Any sudden total blocking of the ureter would cause immediate anuria and therefore no distension at all of the pelvis. Partial or intermittent obstruction may be due to a renal stone formed in the renal pelvis but which has dropped into the ureter, which it partly

blocks. If it cannot pass through, it will more or less plug this tube. Or, the obstruction may be due to a tumor of some other organ which presses on the ureter; or to bands of scar tissue—the result of an abscess or inflammation near the ureter—which, by contracting, pinch across this tube. Or, the trouble may be due to an unusual angle at which the ureter leaves the pelvis, or to an unusual position of the kidney which favors a twist or kink of its ureter. Whatever the cause, if the fluid accumulates intermittently in the renal pelvis, it distends this and its calyces. If the obstructions are frequent and the pressure which develops is high, this causes, in time, atrophy of the kidney, which may become spread out into a thin cyst-like shell. If, however, the pressure of the fluid is less of a factor, the kidney itself may not suffer much, while its dilated pelvis may almost fill the whole abdomen. In cases of hydronephrosis the patient may scarcely know of his trouble, or may have pains which are severe only when the fluid in the pelvis is under pressure.

The treatment is to find out the cause of the obstruction, and, if possible, to remove it. Sometimes the urine must be drained by catheter or by operation. If the cause is a tumor, or bands of adhesions, their removal is indicated. If the cause is a "floating kidney," this may be supported by a proper pad and binder, or, if necessary, may be suspended by operation.

Pyelitis is a condition which deserves especial attention because of the importance of its frequent yet often unrecognized attacks. The word pyelitis means "inflammation of the pelvis of the kidney." The walls of this pelvis are thin and are lined internally with a mucous membrane which continues down the ureter to the bladder. Inflammation of the mucous membrane of the pelvis is pyelitis (pyonephrosis, if this sac is distended). Usually the condition affects but one kidney, rarely both.

When inflamed this mucous membrane swells, its surface cells peel off, its blood vessels become congested, and pus pours out into the pelvis. Often tiny blood vessels rupture

into the sac. When the disease is chronic, practically all of this mucous membrane is destroyed and then the pelvis is like any other abscess. Later, the ureter may become plugged, and then the pus sac may become distended to large size (pyonephrosis). In such cases the pyramids flatten out, the kidney tissues atrophy, and later become only a part of the wall of the sac. Such an abscess may rupture, or the pus within it may dry down to a clay-like mass in which lime salts are deposited till the whole is a mass of almost stony hardness.

In pyelitis the urine at first is cloudy because of the abundance of epithelial cells, mucus, pus, and blood, and also, when its reaction is alkaline, because of the phosphate precipitate (page 281). When the cause is tuberculosis, however, we have an acid pyuria. Some patients with pyelitis may on one day void a perfectly clear urine, and no kidney trouble is suspected; then, on the next day (often the day when it is not examined) it may consist of pure pus. The explanation of this is that, on the days when the ureter from the diseased pelvis is blocked, only the clear, normal urine from the good kidney reaches the bladder. On the days when the pus escapes from the diseased side, however, the diagnosis is easy enough. Should one kidney be entirely destroyed by disease, the other kidney increases in size and does the work of both.

The causes of pyelitis are many. Perhaps an irritating drug like turpentine can inflame the pelvis, but this is doubtful. In nearly all cases germs, especially Bacillus coli (but also Bacillus tuberculosis and the pyogenic bacteria), are the cause. One must remember that bacteria (and some of them quite virulent ones) frequently get into the body and are carried around by the blood stream to the kidneys. As a rule the kidney itself suffers little, unless its own resistance is poor, and then these germs may cause local renal trouble. The kidney's resistance may be lowered by various conditions: general poor health, fevers, as typhoid, simple muscular over-exertion, a cold, some local renal trouble, as Bright's disease, a stone in its pelvis, a local area of tuberculosis on

one of its pyramids, a renal cancer, a twist in the ureter, a tumor pressing on the ureter from the outside, and, possibly, an irritating drug. In another very important group of cases an inflammation starts in the bladder and travels up the ureter to the pelvis. Pyelitis is most common in young girls and in pregnant women.

The symptoms of pyelitis vary greatly. Often they are spectacular, with high fever, a high leucocyte count, and severe chills. In others there may be pain in the back due to the distension of the pelvis, but this is rare. In still other cases, however, there are no symptoms; this is true especially of those cases which complicate a fever, for they merely make the patient a little sicker than before.

In those cases of pyelitis which are not a complication of a pre-existing fever, the patient will have failing health, he becomes pale, loses weight and strength, does not feel well, and has some fever. Such a case may be treated throughout its whole course for typhoid fever, because the patient looks "typhoidal"; others, for malaria, because they have repeated severe chills and fever with profuse sweats; others are told they have an "internal abscess," because they have a hectic fever without sweats; this patient "did have a little fever, probably 'bilious' fever, but now he is all right," yet even now an active pyelitis may be in progress.

The diagnosis is made from repeated, systematic examinations of the urine. Even though the urine may seem normal, the doctor should be sure that both ureters are open. If, on cystoscopic examination, no urine is seen to come from one, a catheter should be passed up that one into the renal pelvis to make sure that there is not a pocket of pus there. No casts will be found. In all cases of pyelitis the urine should be examined for **tubercle bacilli,** and an X-ray picture be taken to see if the renal pelvis **contains a stone.**

The treatment of pyelitis will depend on the cause. If no stone is present and there is no stricture of the ureter, the patient is put to bed, the bowels kept open, a milk diet prescribed and abundant water ordered which often will

wash the condition away. **Hexamethylenamine** (urotropin) is given. In the case of women, especially, the renal pelvis can be repeatedly irrigated. The cases which are complications of acute fevers usually get well without treatment. For those cases due to stone or stricture of the urethra an operation may be necessary. If let alone, such a case of pyelitis may subside, may even get well—though with the destruction of one kidney; but as a rule the renal abscess becomes the centre of a general infection of the whole body.

Renal calculus. As mentioned on page 257, the kidneys are to eliminate many organic substances, as well as minerals, in the urine. Normally, in the urine these are in solution, but under some conditions **one or more of them may separate out and form concretions** (renal calculus, nephrolithiasis) of considerable size and hardness. Some of these kidney stones are made of uric acid, others of oxalate of lime, or of phosphates, etc. Mineral renal calculi usually are very clearly seen on X-ray plates; the organic, not. Why stones form is not well understood, but evidently a nucleus is necessary. Bacteria and mucus can furnish such nuclei. There must also be stasis of urine in the renal pelvis, and conditions are particularly favorable when the urine is concentrated. Some stones, the "coral," "dendritic" (tree like), stones fill the entire pelvis and all of its calyces. Such, since unmovable, may cause much backache, but no colic. Those which are the size of a pea or a little larger may be swept into the ureter and passed in the urine. Some patients have passed several hundred such stones during many years, and suffered terrible colic each time. Many others (about the size of the head of a pin) are passed which are too small to cause pain. Renal stones are most often found in the young and the old, and are much more common in some localities than in others. Budapest is noted for the number of children who have these stones. Some counties in England are called "stone counties" because so many persons are thus affected.

The effects of the stones are various. Their very presence almost inevitably causes infection of the renal pelvis, and

perhaps of the kidney also. A stone may lie quietly in the pelvis of the kidney and give no pain, but usually it does cause a dull backache, for which some other cause is assigned; occasionally it causes severe paroxysms of pain, and usually some hæmaturia. **Pyelitis** with all its symptoms is a natural result. The natural but late result of a large dendritic stone is atrophy of the kidney, which may become only a mass of scar tissue. A very small stone which, because of some change in the position of the body, drops into the ureter may be swept along by the current of urine and be passed without pain. One too large to pass may block the ureter; if intermittently, **hydronephrosis** will develop; if completely, anuria. The result of complete obstruction will depend on the other kidney, for, without operation, the kidney of which the blocked ureter is the outlet is doomed. If, in such a case, the other kidney is diseased, the outlook is bad; if not, it is good. In about one half of all cases however, the renal calculus, if about the size of a pea, slowly makes its way down to the bladder, causing the well-known **renal colic.** This journey, which seldom lasts less than an hour, and sometimes a whole day, starts with a sudden, agonizing pain in the back, usually on the affected side but sometimes on the other. This pain runs down the side, usually to the genitals, but sometimes to the inner side of the thigh. It is terrible in intensity, the worst pain of all, some say. This pain continues until the stone stops in the ureter or it ceases suddenly, when the stone has entered the bladder. With it there may be chills and high fever, often violent vomiting, profuse sweating and even collapse. The patient has an incessant desire to urinate, and passes a scanty, bloody urine (a severe hæmaturia is unusual) which contains also some pus.

The behavior of the other kidney during the colic is important. Sometimes it ceases to excrete urine, a "reflex anuria," supposed to be due to the nervous control of the kidney. Sometimes it excretes great quantities of dilute urine, a polyuria also attributed to reflex nervous influence.

During the colic, hot applications to the affected side are

often very agreeable, also hot drinks, and especially a hot bath. Usually maximal doses of morphia and even chloroform are necessary.

Between attacks, the patient should, above all else, eat very temperately, remembering that the constituents of stones are the ashes of meat, and should drink large quantities of water. There is a great amount of humbug about the best waters to advise, and about drugs guaranteed to dissolve stones, but both medicinal waters and drugs are, so far as we know, all practically useless. Unless the stones are passed readily, by far the best treatment is their surgical removal.

The kidney may be the seat of **various tumors,** some of them malignant. A few are true cancers, but by far the most are hypernephromata, the origin of which are congenital remnants of adrenal tissue in the kidney, which grow to enormous size and which give rise to early metastasis, especially to the lungs. **Huge renal sarcomas** develop especially in young children. Some are congenital and by their size interfere with normal delivery.

The patient with a renal tumor early feels the lump, and suffers dragging pains in the affected side. When the tumor is malignant, **hæmaturia is very common.** In fact, blood in the urine not easily explained by some evident disease should always suggest tumor, of which it is by far the most common first symptom. Anæmia and cachexia come later.

Cystic disease of the kidney (congenital cystic kidney) probably dates back to infancy, although it may not be evident till adult life. Both kidneys are affected and may be so huge as almost to fill the entire abdomen. Usually, however, one is much larger than the other; indeed, in the majority of cases only one is palpable. The natural shape of the kidney is lost, and each feels like a nodular tumor made up of a mass of cysts. The most of them are minute but some are of the size of a bean. The kidney tissue itself, in the adult, is the seat of an advanced chronic interstitial nephritis, with all the symptoms and signs of this condition including high blood pressure, and with hæmaturia, in addition.

CHAPTER XIV

Diseases of the Nervous System

Even a superficial understanding of the organic diseases of the nervous system requires a knowledge of anatomy so accurate that, in these chapters, only a brief discussion of a few of the familiar diseases is possible, and of these we shall mention only their most prominent symptoms.

The Cerebrospinal Fluid

The central nervous system consists of the **brain** and **spinal cord.** These are safely enclosed in bony cases, the brain in the cranium, the cord in the spinal column. They are wrapped up in three membranes, or **meninges** (see page 459), between which is a layer of fluid, a watery pad, which affords the brain and cord considerable protection. This very clear watery **cerebrospinal fluid** is secreted in the ventricles of the cerebrum. It flows out into the subdural (subarachnoid) space through small canals (the foramen of Magendie and the foramina of Luschka). This fluid fills all the cavities within the brain and medulla and surrounds as well the entire surface of the central nervous system. It is some of this fluid which we obtain by lumbar puncture, that is, by the insertion of a hollow needle into the spinal canal between the fourth and fifth or the third and fourth lumbar vertebræ, and the examination of which is so important in diagnosis.

For lumbar puncture a long, strong and pointed needle with stylet is necessary. The needle must be perfectly sterilized. If possible, the patient, when all is ready for the puncture, sits up astride a chair facing its back, his spine bowed well forward. If too sick to assume this posture, he lies on his side in bed, his knees bent, and his back arched forward as much as possible. The entire back is well scrubbed with soap and water, then painted with tincture of iodine as though for an operation. The proper space to enter between the lumbar spines is the one just on a level with a line connecting the crests of the ileums. To indicate this space, the nurse,

when the doctor is ready to insert the needle, places her hand on this crest. The skin at the point to be punctured is first anæsthetized locally by freezing it with ethyl chloride, or the patient, if in bed, may be given a few whiffs of chloroform. The needle is inserted in the midline between the spinous processes at an angle such that will not strike either spinal process. After it penetrates for about three inches, it can be felt to pierce the dura. Then the stylet is removed to see if the fluid will flow. If it does not, the chances are that the dura has not been pierced but has been pushed to one side. There is no danger of touching the cord for it does not reach down to this level. A little fluid, **not over 5 c.c. in all, on the first puncture,** is slowly collected into several test tubes, the needle withdrawn, the hole covered with sterile cotton, and the patient kept in bed for about twenty-four hours **with his head low.** This is done by raising the foot of the bed on blocks. If the patient gets up and about too soon, a severe headache may result. The headache may be relieved by a hypodermic of pituitrin. At later punctures, provided the case is not one of brain tremor, more fluid may be collected. **The cell count** of the fluid is made at once by using a special counting chamber. A count of from 1 to 6 cells per cmm. is normal, from 6 to 10 per cmm. doubtful, and of 10 per cmm. or over is definitely pathological. The rest of the fluid, after the count, may be kept in the ice chest until tested. The tests for **globulin** and **glucose,** the **Wassermann test,** and the **colloidal gold tests** are made as soon as convenient. The character of this fluid in the various diseases will be mentioned later.

The Brain

The brain consists of cerebrum, cerebellum, pons, and medulla. It weighs about 1500 Gm. (three pounds).

The cerebrum is the largest portion of the brain. It consists of two halves, of almost equal size, united at their lower borders by the pons, or bridge. Each half, or hemisphere, of the cerebrum is hollow, and the cavity, the **lateral ventricle,** is filled with the cerebrospinal fluid, which is formed here.

When, because of malformation or disease, the canals which connect the ventricles of the brain with the subdural space are closed, and there is, therefore, no outlet to the flow of the ever-forming cerebrospinal fluid, **internal hydrocephalus** develops. If the patient is a baby, the bones of whose skull have not yet united, each of the lateral ventricles (the ones within the hemispheres of the cerebrum), instead of containing about 50 c.c. of fluid may hold 500 c.c., or more. The hemispheres thus distended resemble thin bladders, and the head is swollen to huge size. This terrible condition is named

Fig. 106.

Fig. 107.

Fig. 106.—A cross section through a tip of the brain showing the arrangement of the gray and the white matter. The gray matter is external and near the ventricles, the white is internal.

Fig. 107.—Cross section of the spinal cord showing the arrangement of the gray and white matter. The gray matter is internal and the white is external.

hydrocephalus or water in the brain. A somewhat similar condition occurs in adults as the result of inflammations; but, since the skulls of adults cannot distend, the pressure of a very little extra fluid causes not only severe symptoms such as headache, vomiting, blindness, but even death in some cases.

The cerebrum consists of gray matter, which is spread on its surface in a thin layer about one eighth of an inch thick called the **cortex** (*a*, Fig. 106), and of the **white matter** (*b*) which lies between the cortex and the ventricles (*c*). The gray matter is the important part; this contains all the **brain cells,** the batteries of the nervous system. The white matter consists almost entirely of long **fibres,** the wires which con-

nect the cells with each other and with the organs, the muscle fibres, etc., which these cells control. These fibres are so fine that a strong microscope is needed to see them, yet some of them are fully two feet long. If the cerebrum had a smooth surface there would not be cortex enough, and so

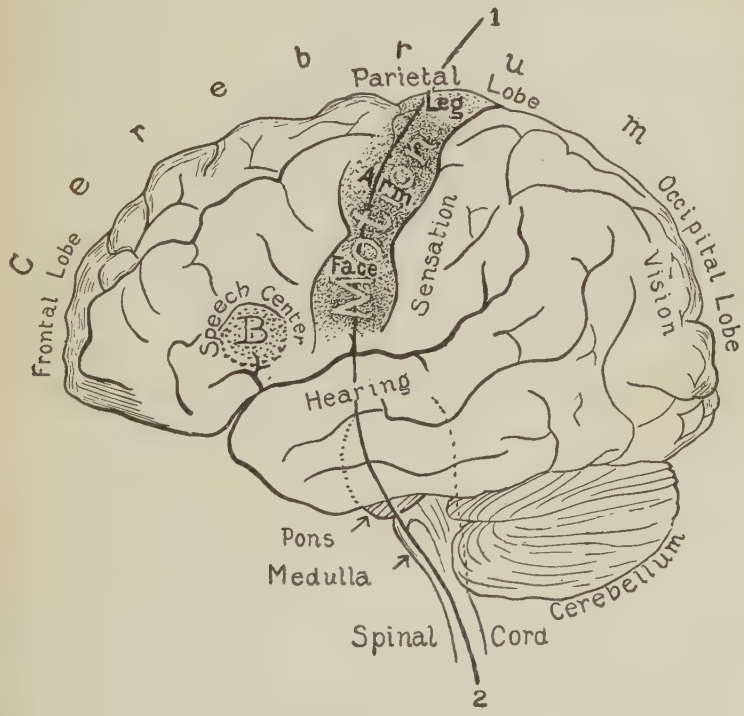


FIG. 108.—Side view of the brain. The line 1-2 is the direction of the schematic cross-section represented in Figs. 109, 110 and 116.

its surface is folded in many convolutions with deep crevices, or fissures, between. In this way, the amount of gray matter is considerably increased without increasing the size of the brain. **The larger convolutions** are fairly constant, so that they serve as excellent landmarks in finding the various areas of the brain.

The brain cells of the cortex look alike, but have very

different duties to perform. There is, for instance, on each hemisphere a vertical band of cortex, the **motor area** (Fig. 108), which governs the voluntary movements of the muscles of the opposite half of the body. This can be accurately mapped out. We know the exact spot where lie the cells in which originate the voluntary movements of the muscles of

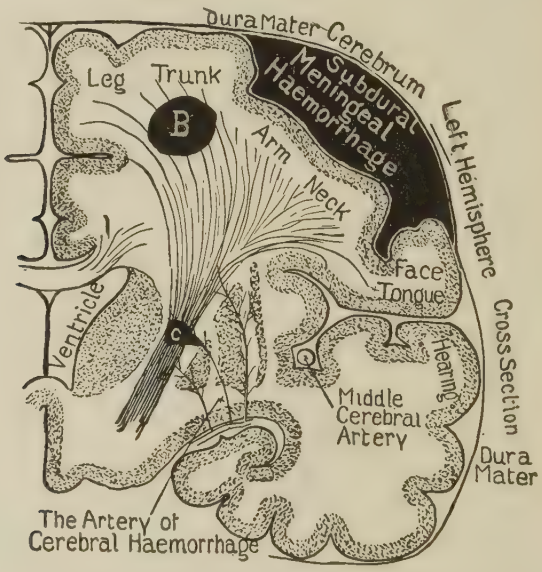


FIG. 109.—A cross-section of the left hemisphere of the brain through the motor area. The subdural meningeal hæmorrhage compresses the brain over the arm-neck area. There is no real destruction of brain substance, but paralysis would result from pressure. *B*—Subcortical hæmorrhage. This hæmorrhage does destroy brain substance. Although a smaller hæmorrhage than the subdural, it produces possibly more paralysis, since it cuts fibres which have converged somewhat. *C*—Hæmorrhage into the internal capsule. This explains the common "stroke" of paralysis. Although the hæmorrhage is small, it causes total paralysis of the right side of the body, since it cuts all the fibres in the internal capsule.

the face, the thumb, the hands, the arm, the trunk, the leg, etc. If we wish to move a muscle, it is these particular cells which must send the stimulus down along their fibres. If we stimulate these cells with an electric current, the muscles which they control will contract. If, therefore, a man has paralysis of certain muscles, and this paralysis is due to disease of his cortex, we know, before we open the skull,

almost the exact spot on the cortex where we shall find that trouble; or if, as sometimes occurs after fractures of the skull, the patient has convulsions of certain muscles, or convulsions of the whole body which always begin in certain muscles ("Jacksonian epilepsy"), we can tell just where to look for the sliver of bone, etc., which is pressing on the cells that govern these muscles.

Though the motor area of each hemisphere is large, the fibres from it converge and pass out of that hemisphere into the pons collected into a small bundle, the **internal capsule.** This bundle then passes through the pons into the cord, crossing, as it does so, the bundle from the other side. It runs down the cord as the **pyramidal tract** (Fig. 109). A very small injury to this bundle will paralyze many more muscles than will a much larger injury to the cortex itself. For illustration, the brain is like a telephone station, where one blow of an axe can sever all the wires at the point where they leave the building, but a similar blow on the switchboard would sever only a few. The ordinary cause of a stroke of apoplexy followed by paralysis of one half the body (hemiplegia) is usually a tiny hæmorrhage from a blood vessel in this capsule. A very much larger hæmorrhage nearer to, or at the cortex, might paralyze one limb, but hardly a whole half of the body. Hemiplegia may be due to the rupture of a miliary aneurism on a tiny artery running to the internal capsule, the **artery of cerebral hæmorrhage** (see page 90), or to the plugging of this artery by a thrombus or embolus, and the subsequent death of the fibres which it supplies with food. Why this artery, which is of the greatest importance in the brain, should suffer so much oftener than do any of the others, we do not know.

Immediately after a shock, one whole half of the body, as a rule, is paralyzed. Then, gradually, the person recovers the use of certain muscles, usually those of the leg, often those of the upper arm, least often those of the hand. The reason for this is that, while the hæmorrhage may actually destroy the fibres of only a few muscles yet it injures temporarily all of those in its neighborhood, perhaps by the pressure of the

escaped blood, or by the œdema which surrounds it. As the swelling from the hæmorrhage diminishes, these latter fibres will resume their function; but those destroyed will never do so.

In **Broca's convolution** (Fig. 108 *B*), the cells look like those of the motor area, but really have nothing to do with

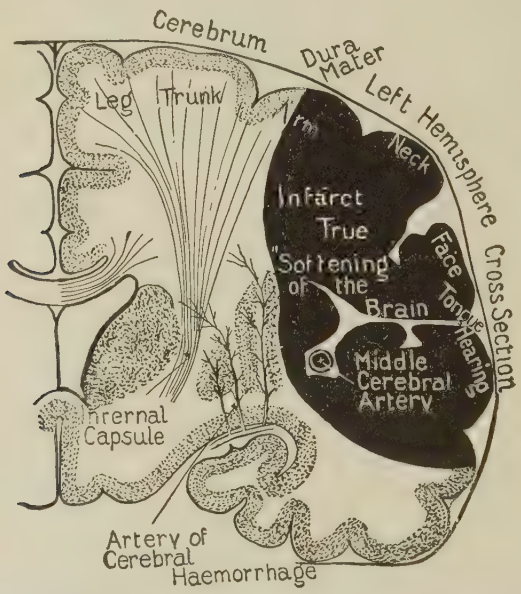


FIG. 110.—A cross-section of the left hemisphere of the cerebrum through the motor area. The middle cerebral artery is represented as plugged by a thrombus. That part of the brain (black area) supplied with food by this artery has died (infarction, or "softening"). This patient would have paralysis of the right side of the face, tongue and neck, and part of the right arm; he would be unable to talk and understand what was said to him.

muscles. Broca's convolution is an area on the left hemisphere, about a square inch in size, which adjoins the motor area. Here are the cells in which are stored up the **combinations of muscular movements** necessary to speak each word. They are not the cells which govern the muscles of speech; these cells are in the motor area itself. Each word, even the simplest, requires for its utterance quite a combination and sequence of combinations of muscular contractions; not only

must the muscles of the vocal cords contract, but also those of the throat, of the tongue, of the soft palate, of the lips, and of the chest wall. To pronounce even a short word is a very complex action. In the cells of Broca's convolution, these combinations are stored. They direct the cells of the motor area, and these make their muscles contract at the proper time and with the proper force. This area (Broca's convolution) is so near the left motor area that disease of the latter often affects the former. This is why so many persons who are **paralyzed on the right side** (due to a lesion of the left hemisphere) **are unable to speak;** while those paralyzed on their left side almost never have any speech disturbances. In some cases they do, but these usually are left-handed persons whose speech area is on the right hemisphere. (This is one proof that to be right-handed or left-handed is not a matter of training, nor of habit, but depends on the very structure of the brain itself. Lose the right hand, and, if right-handed, the left hand may become fairly proficient, but never entirely so).

When Broca's convolution is destroyed, the person is said to have **motor aphasia.** He understands all that is said to him; he knows the words he wants to say; he may write the word with the hand which is not paralyzed; he can read it; he has absolutely no paralysis of the vocal cords; but, for a few days at least, he cannot produce the sequence of movements necessary to produce that word; and if he tries, he makes an unintelligible noise. During that period, he is somewhat like the banker who has forgotten the combination of his safe. He may turn the dial as he will, but if he does not turn it back and forth each time to exactly the right figures the safe will not open. The lock is all right, and the man is strong enough to turn the dial any number of times he will—the trouble is that he has forgotten the combination. To cause a permanent motor aphasia, however, the lesion must (and it usually does) affect the white matter just beneath this convolution where are the fibres going to and coming from other parts of the brain. When only the

gray matter of Broca's convolution is destroyed, the aphasia is transitory.

In other areas of the cortex are stored up the memory of the way the words sound which we want to speak. Injury here causes **sensory aphasia.** This patient may be able to talk volubly, but point to a chair and ask him what it is, and he cannot remember the word chair, although he may use that word frequently in his rambling conversation. Ask him if it

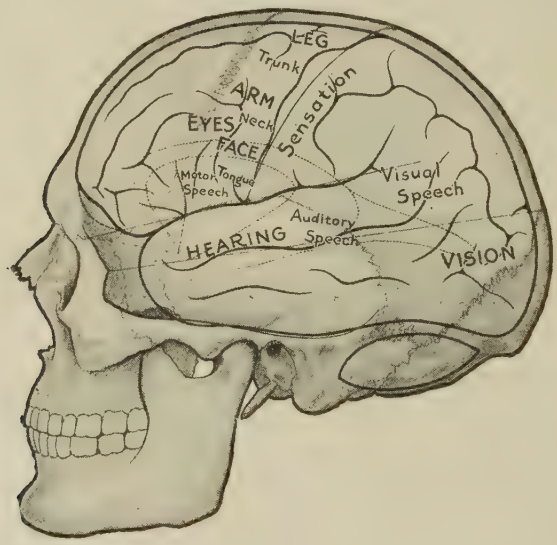


Fig. 111.—Landmarks of cortex.

is a chair and he at once assents. There is no paralysis, and the word is correctly spoken; that is, it is sensory and not motor aphasia. There is another cortical area in which is the memory of the way words look when written. If this area is injured the person has **alexia,** (no reading ability).

We are all functionally aphasic at times. I meet an old acquaintance, but cannot recall his name, try as hard as I will. I know his name well, but cannot remember it. I have, for an instant, sensory aphasia.

True brain disease may cause an aphasia which is total at first, but which in time partially, or entirely, passes away.

In such a case, the actual destruction of cells and fibres was perhaps not as great as was at first imagined, but many cells and fibres were, for a while, rendered functionless, since they lay in the halo of temporary disturbance which surrounds each destructive lesion. In other cases it would seem as though another part of the cortex could take over the work of the destroyed area, for sometimes, by careful training, a man with motor aphasia may learn to talk again.

There are definite areas on the rear of each hemisphere where the **fibres of the optic nerves end.** It is by means of these receiving cells that we see. The eyes may be ever so good, the optic nerve ever so perfect, but if these cells are diseased, we are more or less blind. For illustration, my friend is telephoning to me. He may talk distinctly, his telephone may be in perfect order and the wire intact; but if the receiver I hold in my hand is out of order I may not be able to hear a word. So, although my eyes, my optic nerves, etc., are perfect, if all of both these cortical areas is diseased, I can not see anything; I have cortical blindness. **Cortical blindness** of one optic area (that is, of the optic area of the posterior tip of one cerebral hemisphere), always affects both eyes equally. Total blindness in one eye may be due to disease of that eye itself, or to disease of its optic nerve. Just behind the two eyes, however, their two optic nerves unite, then separate, and continue to the brain as the two optic tracts. In each of these tracts is just half of each optic nerve, so that, if one tract be injured there is complete blindness of exactly one half of each retina. For example, if the right tract be injured the patient is blind on the right half of each retina, so that he with either eye can see nothing to his left, but will see perfectly to his right. This is **hemianopsia or half-blindness.** Destroy the cortical optical area of the hemisphere to which that tract runs and we will have this same form of hemianopsia. In any case of blindness we can thus locate the trouble.

There are, also, cortical areas where we **smell,** others where we **hear,** etc., and larger areas, especially under the

forehead, the office of which is not known, but diseases of which change a man's disposition or moral character.

The importance of the brain cortex is strikingly shown in general paralysis of the insane, or dementia paralytica (page 531).

The cerebellum has functions which are little understood. It is enough to say that disease here causes, among other symptoms, first, motor incoördination, or inability to use the muscles accurately (the patient stumbles and fumbles, is dizzy and staggers); and, secondly, his muscle action lacks tone. It is important in diagnosis that cerebellar convulsions are essentially tonic, that is steadily tense in character, while

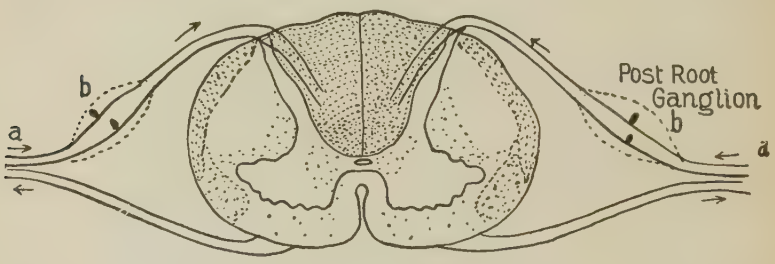


Fig. 112.—Cross-section of the normal spinal cord. The dotted areas show the location of the sensory fibres *a*, which run through the posterior root ganglia *b*, to the cord and up to the brain.

those of the cerebrum are clonic, that is, are very jerky. A patient with cerebellar disease has **a staggering gait,** he lurches from side to side as though he were drunk, his feet wide apart but the steps not long and not stamping. This, the cerebellar gait, is called also the vertiginous, reeling, titubating, or drunken gait of cerebellar ataxia. In these cases, it is the trunk which would seem to be ataxic and the legs which seem to maintain the balance.

The tendency to reel always in the same direction suggests a unilateral cerebellar lesion.

The pons is a connecting link between the two hemispheres. From it also starts the **medulla,** which is the bulbous upper end of the spinal cord.

Let a **tumor** grow anywhere **in the brain** and certain **general symptoms** usually follow. Among these are **headaches** which are most severe in cerebellar tumors, and least so when the tumor is at the base of the brain. These headaches are severe and persistent, boring or stabbing, with exacerbations of great severity. Another symptom is **local tenderness of the scalp** accompanying these headaches; and **vomiting,** usually without any nausea (cerebral vomiting), worst in cerebellar tumors, and especially so if they are rapidly growing. Other general symptoms are **vertigo** and annoying **paræsthesias.** Among the general physical signs are changes in the retinæ of the eyes which may result in blindness, due to pressure on the optic nerves producing also the so-called choked disc, which is the most important and most common of all signs of brain tumor. Other more local and more definitely localizing signs (that is signs which indicate where in the brain the tumor is) are: **paralyses;** irritation of the cortex causing **convulsions** which begin with certain muscles; changes in the **special senses,** etc.

The Cranial Nerves

The central nervous system communicates with the rest **of the body through the peripheral nerves,** which are bundles of the white fibres, Fig. 112, *a*. Nerves are always arranged in pairs, one on each side of the body. Some nerves, called **motor nerves,** run from the brain to the muscles. Cut these, and the muscles they supply are paralyzed. The cells from which these fibres start are in the central nervous system. Other nerves, called **sensory nerves,** run from the skin and sense organs to the brain; cut these, and the area of skin, etc., from which they come is made insensible. The cells of these sensory fibres are outside the central nervous system, collected in small masses called ganglia (Fig. 112, *b*). Most peripheral nerves are **mixed,** that is, they contain both motor and sensory fibres.

Connected with the brain are twelve pairs of nerves, called **cranial nerves.** The first pair are the **olfactory nerves** or nerves of smell, which connect the mucous membrane of the

nose with the brain; the second, the **optic nerves,** or nerves of sight, which connect the retinæ of the eyes with the brain. The third pair, the **oculomotor nerves,** are motor nerves which supply all but two of the muscles which move the eyeballs; the fourth (trochlearis) pair, the **trochlear nerves,** supply one of these two (the oblique muscle); and the sixth pair, the **abductor nerves,** the other of these two muscles (the one which rotates the eyeball outward) not supplied by the third pair.

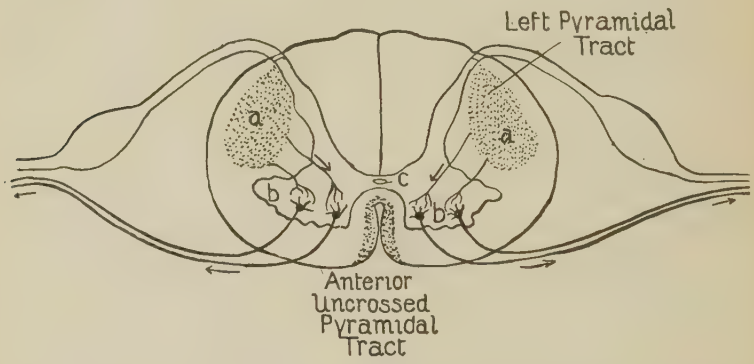


FIG. 113.—A cross-section of a normal spinal cord. The dotted areas represent the location of the motor fibres which run from the cortex of the brain to the motor nerve cells which control the muscles. *a*, pyramidal tracts of motor fibres; *b*, anterior horn cells, the nerve cells which directly govern the contractions of the muscles.

The fifth pair, the **trigeminus nerves,** are the largest of the cranial nerves. The trigeminus nerve is a mixed nerve whose sensory portion comes from the skin of the head and face, the outer membranes of the eyes, the mucous membrane of the mouth, and the pulp of the teeth, and whose motor portion innervates the muscles of mastication. Each fifth nerve divides into three large branches. The first branch (supraorbital) supplies the tear glands, the membranes of the eye, the skin of the forehead, and the inside of the nose. The second branch (infraorbital) supplies the skin of the nose, cheeks, upper lip, lower eyelid, and the upper teeth. The lower branch (mandibular) contains the motor portion and also receives sensory fibres from the skin of the temple,

lower lip, lower jaw, part of the tongue, and the teeth of the lower jaw.

This fifth nerve is the nerve of **facial neuralgia,** or tic douloureux. In this disease the whole nerve may be affected, or one branch only. Which, the patient knows only too well. Mild cases of facial neuralgia may start in a decayed tooth, or be due to some other local disease. Remove this, and the patient at once gets well. True "tic douloureux," however, is due to disease of the ganglion of this nerve. This is an affliction of middle life especially. In a severe case the pain comes on in paroxysms of terrible intensity, separated by periods of remission which may last from minutes to months, but which grow shorter as the disease progresses. The paroxysms are brought on by cold, by movements of the face or tongue, by a touch on the skin, in fact by almost any stimulus to the head or face. The pain often starts at one spot and radiates along the branches of this nerve, and even into those of other nerves. It is so frightful that it sometimes leads to suicide. The cause is probably degeneration of the Gasserian ganglion, the result, perhaps, of hardening of the arteries which run to it. As medical treatment, one may try nitroglycerine, or strychnine in large doses (begin with 1/20 gr. hypodermically, three times a day, and increase slowly until the dose is even 1/5 gr., each entire course to last six weeks). The injection of the branches of the nerve by alcohol, and the evulsion of a branch are common practices. For the latter treatment the branch of the nerve is exposed at the point where it is nearest the skin, is seized by a pair of forceps, and as much of the nerve as possible torn out. The nerve will grow again in time, but the patient sometimes has, as a result of this operation, about eighteen months or more of comfort. Then an inch or so more may be torn out at the same place. While a few cases have recovered spontaneously, and while the above minor operations may give relief, yet the best treatment of tic douloureux is the **removal of the root of the Gasserian ganglion** by an operation within the skull. Then, the cure is permanent. After this operation

the nurse has considerable responsibility, for part of the patient's face is now insensible, and hence must be carefully guarded from all injuries until the tissues readjust themselves to their new state. The eye especially needs watching, since the patient will not know when dust is on the cornea, and this may start up an inflammation which may even destroy the eye. To prevent this the patient for a while wears a shield over that eye, and the nurse at regular intervals washes the conjunctival sac out with some mild wash.

The seventh pair of cranial nerves, the **facial nerves,** run to practically all the muscles of the face. When one of this pair is much affected facial paralysis (Bell's palsy) is the result. This paralysis may be due to trouble anywhere along the nerve from brain cortex to the muscles, but most often the trouble is in that part of the nerve which lies in a long canal in the temporal bone. This canal is very close to the middle ear, and if this is inflamed the disease easily affects this nerve also. The commonest cause of facial paralysis, however, is to catch cold in the face by exposure to a strong wind, etc. The nerve becomes inflamed (neuritis) and swells. For the nerve to swell where it lies under the skin does little harm, but for the swelling to extend to the portion lying in this canal of hard bone, which is only just big enough to hold a normal nerve, is more serious, for the pressure produced paralyzes it. In a case of facial paralysis the affected side of the face is smooth, and does not move when the patient frowns, closes his eyes, or moves his mouth, etc. The upper eyelid droops, yet the patient cannot close his eye. The corner of the mouth is drawn up and moved to the good side, since the paralyzed muscles are now limp and do not normally oppose their antagonists of the other side of the face. In eating and drinking the food drops from the paralyzed corner of the mouth. Ask the patient to whistle and he cannot. The cases due to cold practically always get well in time. If seen when almost well, one may mistake which side was paralyzed, for now, for a while, the muscles formerly paralyzed contract more strongly than do the normal ones and

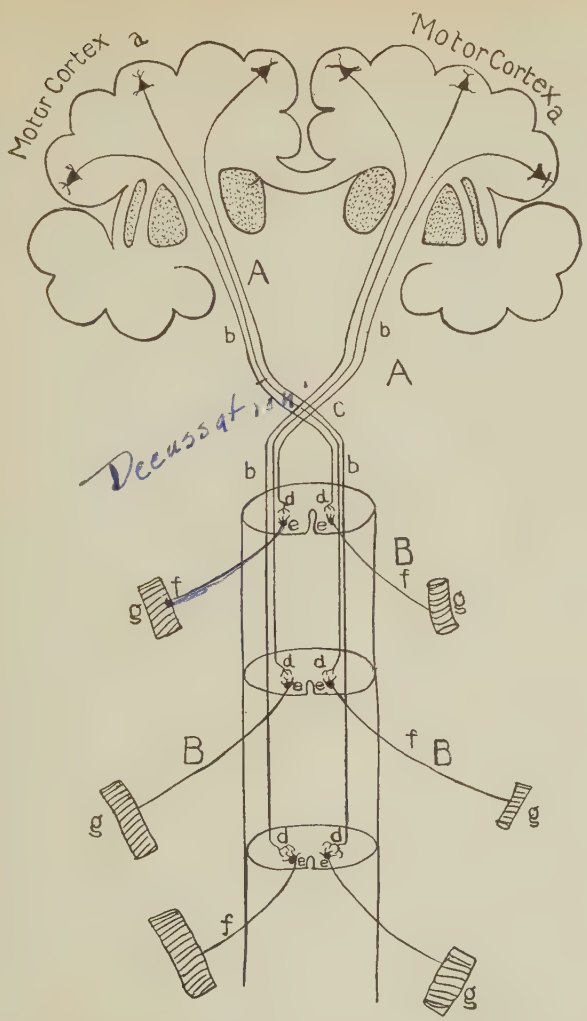


FIG. 114.—Scheme of the motor path. *A*, upper motor neuron; *B*, lower motor neuron. *a*, motor cortex, containing the cells of the upper motor neuron; *b*, fibres of the upper motor neuron, which run down the cord and end in the anterior horns, *d*, in contact with the anterior horn motor cells, *e*; *f*, fibres of the anterior horn cells, *e*, which run to muscle fibres *g*. *c*, decussation of pyramidal tracts.

so wrinkle especially the side which formerly was smooth. The treatment of facial paralysis is to remove the cause if possible; e.g., to treat the ear trouble. If the paralysis is due to cold, little can be done, save to keep the paralyzed muscles in as good condition as possible by electricity, using, during the second week, for five minutes at a time a galvanic current strong enough just to contract the muscles; after two weeks, massage is prescribed. At the onset, a purge is given and a blister is applied over the exit of the nerve. Salicylate of sodium, also potassium iodide, are favorite remedies.

Each nerve of the eighth pair has two portions, the **auditory (cochlear) portion** and the **vestibular portion.** The former is the auditory nerve, which connects the cochlea of the inner ear with the brain. Disturbances of this nerve impair hearing. Disturbances of the vestibular nerve produce vertigo (a sensation of turning or falling) and nystagmus. Ménière's syndrome is an acute disturbance of a mechanism of which this vestibular nerve is a part. These attacks begin suddenly with buzzing in the ears, vertigo, staggering, nystagmus, sweating and vomiting, and sometimes unconsciousness. They usually recur until complete deafness is reached. For treatment, one may use quinine, or sodium salicylate (gr. 5).

The ninth pair are the **glosso-pharyngeal nerves,** the nerves of the sense of taste and the motor nerves of some of the muscles of the throat.

The tenth pair are the **vagi,** or **pneumogastric nerves,** the motor nerves of the voluntary muscles of the throat and larynx, and the nerve through which the central nervous system slows the rate of the heart beat. Branches go also to the lungs, but their function we do not know; others to the stomach and œsophagus, which are important in vomiting at least. They may have other, but not well-understood functions. If one vagus alone is cut there is unilateral paralysis of the palate, pharnyx, and larynx, as a result of which swallowing is difficult and the voice is nasal. Paralysis of

one vagus may disturb temporarily the cardiac rhythm, but complete paralysis of both vagi is followed by tachycardia, since then the accelerator nerves of the heart (sympathetic fibres) lack the normal inhibition of the vagi.

The eleventh cranial pair are the **spinal accessory nerves,** so named because one branch of each joins the tenth nerve of that side. The other branch is the motor nerve of the sterno-mastoid and trapezius muscles. The sterno-mastoid is the conspicuous muscle of the neck which runs from just behind the ear obliquely downward and forward and attaches to the collar bone and sternum. The trapezius is the large

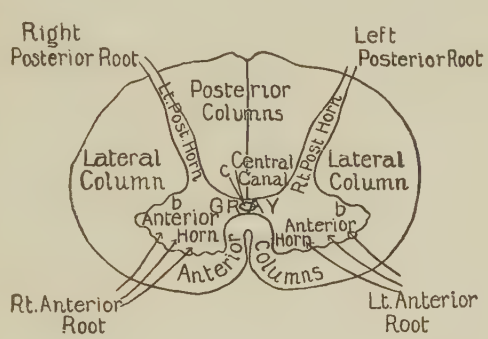


FIG. 115.—Cross-section of the normal spinal cord.

heavy muscle which runs from the base of the skull down the back and whose edge determines the curve of the neck and shoulder. "Wry-neck" or "torticollis" is due to contraction or shortening of these muscles and is sometimes cured by cutting them. In spasmodic torticollis the head keeps up a series of spasmodic jerks which draw the chin down to the shoulder. In this disease often other deep neck muscles also are involved. The treatment of this purely nervous disorder is very difficult. Even radical cutting of these muscles often fails.

The twelfth pair, or **hypoglossal nerves,** are the motor nerves for many muscles of the tongue and throat. When one of this pair is paralyzed, the tongue is protruded towards that side.

The Spinal Cord and Its Nerves

The spinal cord, a direct continuation of the medulla oblongata, is that part of the nervous system within the backbone. It is a cord about eighteen inches long and approximately the size of a man's finger. Like the brain, it consists of gray and white matter, but, while in the brain the gray matter is external and the white internal, in the cord the gray matter is in the centre and is surrounded on all sides by the bundles of white fibres of sensory fibres running up to the brain, and of motor fibres going down from the brain. The gray matter is shaped like two pairs of horns (Fig. 113), **the anterior horn** and the **posterior horn.** The cord gives off thirty-one pairs of **spinal nerves.** Each is formed by the union of two roots, **an anterior or motor root,** and **a posterior or sensory root,** on which is the sensory ganglion. These two roots unite to form one spinal nerve. As a result, the spinal nerves all are "mixed." Those leaving the right side of the cord supply the muscles, skin, and organs on the right side of the body; those of the left side, the corresponding muscles, etc., of that side of the body.

On page 316 we described the **motor area** (Fig. 108) of the brain cortex where are the brain cells which generate the nervous impulses by means of which the will makes a muscle to contract. Fibres of these cells of the motor area pass through the medulla to the cord in a bundle of motor fibres, called from its shape the **pyramidal tract** (Fig. 114, *b*). In the medulla this bundle or "tract" crosses that from the other side, *c*, and then each travels down the cord on the opposite side from that on which the cells lie, but on the same side as the muscles for which their impulses are intended. There is another bundle of fibres from the motor cortex which do not cross at the decussation of the pyramids. This is called the **direct pyramidal tract,** and yet every one of its fibres does finally cross to the other side, but just at the level of its termination. At intervals along the cord, (*d*), individual motor fibres, those of the pyramidal and those of the direct tracts, leave the white matter of the cord, **enter the gray matter of its anterior horn,** and come to an end near a group

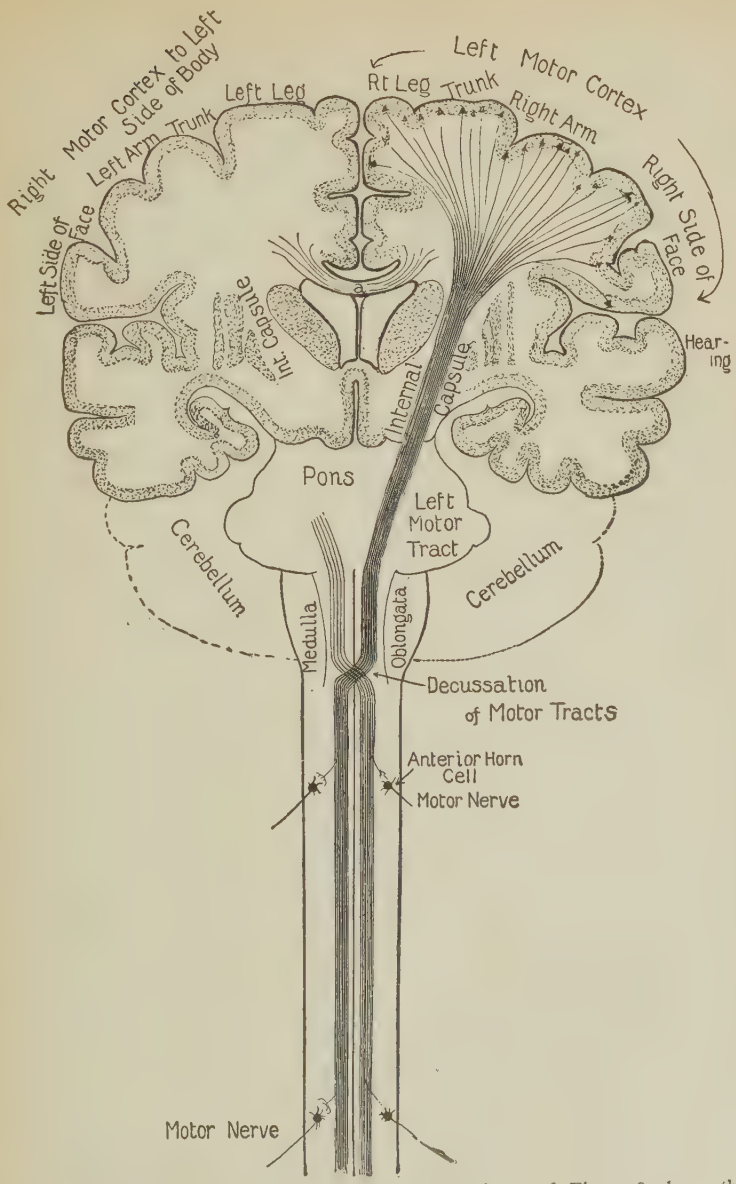


FIG. 116.—Diagram of the motor tract. The line of 1-2 of Fig. 108 shows the direction of this schematic section of the central nervous system.

of motor cells. They never pass out of the cord into a spinal nerve. All of the motor fibres of the spinal nerves are fibres of the cells, *e*, in the gray matter of the anterior horns. These fibres, *f*, run from these cells into the spinal nerve and its branches, each to its own muscle fibre, *g*.

From what has been stated, it is seen that the brain controls each muscle fibre through a **combination of two cells with their fibres.** The cell in the motor area with its fibre running down the cord is called the upper cell or, more correctly, **the upper motor neuron** (Fig. 114, *A*), and the cell in the cord, whose fibre runs to the muscle, is called the lower cell, or **lower motor neuron** (*B*). The motor nerve of a muscle is a bundle of thousands of these fibres, each coming from one motor cell in the cord, and each going to one or a few of the thousands of muscle fibres of which the muscle is a bundle.

Paralysis of a muscle may be due to trouble in either of these kinds of neurons. **Suppose the motor nerve of the muscle** is cut somewhere between it and the spinal cord (Fig. 114, *f*). That muscle will be **paralyzed**; that is, the man cannot through any effort of will move it. Also, it will take no part in reflex movements. But that is not all; **this muscle becomes limp and wastes away,** that is, it atrophies, since its lower motor cells govern not only the contractions, but also the health of the muscle fibres which they control. Soon the injury to a spinal nerve will probably heal. But suppose that, not the nerve fibres, but the **anterior horn motor nerve cells,** from which they come, **are destroyed.** Then the nerve will not grow back and **that muscle will never be useful again.** In anterior poliomyelitis of infants this is just what does happen. (Page 490.)

But suppose it is the **upper motor neuron** which is in some way destroyed. Then a very different state of affairs exists in the muscle. It will be **paralyzed** as far as the "will" goes, but not necessarily for reflex (unconscious) movements, for these originate in the nerve cells in the cord or medulla. It will not atrophy, for the lower motor cells which govern the

growth and health of the muscle fibres are uninjured; it will not be limp but will remain permanently **more tense than normal.** This paralysis seldom affects a part of one muscle, or one single muscle, or only a few muscles, but usually a whole limb, both limbs, or an entire half of the body (hemiplegia).

A good illustration of this form of paralysis is the **spastic** (that is "stiff") **paralysis of those infants** who during birth received some mechanical injury which may have caused the rupture of a blood vessel in the meninges of the brain. The long-continued pressure of the escaped blood may injure large areas of cortex, much more than the motor area, hence these children are usually mentally deficient. Many have convulsions. When such a child should begin to walk, it is noted that the legs especially, and the arms also, are stiff. During life his movements are awkward, stiff and weak. Since those muscles which draw the feet and knees towards each other (the adductor muscles) are naturally stronger than those which spread those limbs apart (the abductor muscles) these persons walk by "cross-legged progression," called also the "scissors" gait. That is, in each step the leg is moved not only forward but is swung around across the front of the other. When both legs and both arms, or both arms only, are paralyzed, the disease is called **diplegia**; when both legs alone, **paraplegia**; and when the arm and leg on the same side, **hemiplegia.**

A more common illustration of upper motor neuron paralysis is the **hemiplegia of adults.** If a hæmorrhage, embolus, or thrombus destroys the fibres from the motor area in the internal capsule, the arm and leg of the opposite side ultimately become stiff and more or less paralyzed, and the reflexes exaggerated. A better illustration still, since in the same case we may compare both an upper and a lower neuron paralysis, is that which follows a **transverse crushing injury to the cord** such as that due to a fracture of the spine or a knuckle in the backbone due to tuberculosis. There will be **stiff ("spastic") paralysis** of all muscles on both sides whose

nerves leave the cord below the crushed spot, and **limp paralysis** of the muscles whose motor nerve fibres come from cells in the crushed place. There will also be **insensibility of the skin below** the crush, since the sensory fibres from below the injury no longer reach the brain. Tumors of the cord ultimately will give almost this same picture, although in the early stages only a part of the cord will be disturbed by the pressure.

Another good illustration of upper neuron disease is seen in adults with **spastic (senile) paraplegia,** a chronic stiffness of both legs due to a gradual degeneration of the fibres in the pyramidal tract. The person so afflicted walks stiffly as though wading through water, the knees always touching each other and hardly raising his feet from the ground.

In the white matter of the cord one can pick out various bundles of fibres. The pyramidal tracts run down in definite portions of the cord (see Fig. 113, *a*). There are definite bundles of fibres which convey sensations from the muscles; others which convey the sensation of touch, pain, heat, cold, etc., for each of these sensations has its own service of cells and fibres. One can, therefore, diagnose the position of some lesions by noting the way they have affected the various sensations. For illustration, here is an adult who has lost the sensations of heat and cold and of pain, but not of touch in the skin of his hands and arms; he has often scalded himself without realizing it; babies have been killed by being bathed in too hot water by these patients. Hearing such a story, one suspects at once a disease which has destroyed the cord only in its centre, and is probably **syringomyelia** (syrinx, pipe; myelia, cord; therefore, freely translated, a cord like a hollow pipe). There is normally a tiny canal down the middle of the cord, Fig. 115, *c,* but in syringomyelia, (Fig. 117), this canal, (*c*), is much larger than normal, not because the canal is distended (hydromyelia), in which case no tissue is destroyed, but because there is destruction of the gray matter around this canal. Since the fibres carrying the thermic and pain sensations cross to the other side of the cord through the

gray matter in front of this tiny central canal, those are the ones which are destroyed in syringomyelia. Nevertheless, the skin of these patients feels touch with normal acuity because the fibres carrying this sensation travel up the cord in the posterior columns at some distance from its centre. There will be other symptoms also in syringomyelia, spastic paralyses with muscular atrophies, pains, lateral curvature of the spine, etc.

Certain fibres which are responsible for **joint sense** (often, but incorrectly, called muscle sense) travel up the posterior

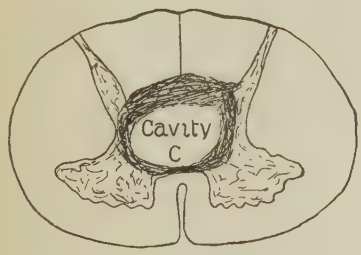


FIG. 117.—A cross-section of the cord of a case of syringomyelia. There has been a growth of the "glia" tissue around the central canal, and a cavity, *C*, forming in this tissue has greatly increased the size of the canal.

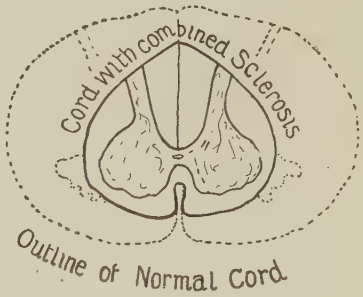


FIG. 118.—A cross-section of the spinal cord of a case of combined sclerosis. The white matter especially has atrophied. The symptoms of this patient might resemble those of a case of locomotor ataxia.

columns of the cord. (There is a muscle sense by which we estimate the weight of objects, but that is a different matter.) **Tabes dorsalis or locomotor ataxia** is due to a form of lues which destroys these fibres (Fig. 119), with the result that the patient does not know where his limbs are (page 533).

Neuritis. The nerves themselves may be the seat of disease. Neuritis means inflammation of a nerve. This may be acute or chronic, and affect a single nerve, local neuritis, or many nerves, multiple neuritis. Local neuritis may follow exposure to cold (e.g., Bell's palsy, see page 326), to injury, or to the extension to the nerve of an inflammation from a neighboring organ (as in lumbago and sciatica due to inflammation

of the joints of the spine). A nerve which is the seat of neuritis is tender when pressed on and always will show evidence of disturbance of its functions; that is, more or less paralysis of the muscles it supplies, trophic changes causing atrophy of these muscles, glossy skin, brittle nails, etc., and various paræsthesias from the skin from which it receives sensory fibres (crawling, burning, tingling, smarting, etc.). If the median or internal poplitæal nerves running down the front of the forearm and legs are affected the neuritis is particularly painful (causalgia).

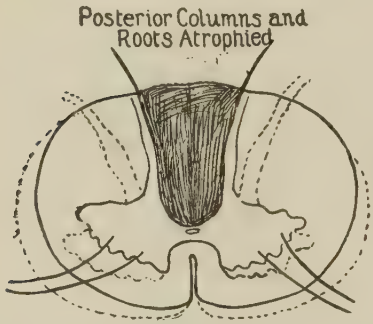


FIG. 119.—Cross-section of the cord of a case of locomotor ataxia. The dotted lines show the outline of the normal cord.

Sciatica is a good illustration of a local neuritis. The sciatic nerve is the largest nerve in the body. Its motor branches supply nearly all the muscles of the legs and feet and it receives the sensory fibres from nearly all the skin of the lower extremities. One form of neuritis of this nerve is due to severe muscular exertion, to the pressure of tumors in the pelvis, and most often to chronic "rheumatism" of the spine in the region where the roots of the sciatic nerve leave the spinal column. The pain of sciatica is intense. It runs from the thigh to the foot, and is made much worse by any motion of the leg which puts this nerve "on the stretch." It is for this reason that the patient keeps his knee bent and walks on his toes. Pressure on the nerve is very painful. There may be wasting of the muscles. Another form of sciatica, the true sciatic neuralgia, more like the tic douloureux, is said to be due to ganglionitis of the lumbosacral ganglia. In these cases the paralyses, atrophies, and trophic disturbance are more marked, and, in the etiology, focal infections, exposure to cold and wetting, etc., are more conspicuous. **Neuritis and neuralgia are not the same conditions.** The term

neuritis is used when we can prove definite inflammation of the nerve itself (tenderness of the nerve trunk, muscular atrophy, paralysis, trophic changes, etc.). Neuralgia (nerve pain) is a term which covers the pains of neuritis but also many other pains in the limbs, muscles, etc., which may be fleeting or migratory. Such usually are pains from inflamed joints or from diseased internal organs (viscerocutaneous reflexes), e.g. the pains down the left arm in heart disease, the aching in the right shoulder blade in gall-bladder troubles, etc. While the pains of a true neuritis may vary greatly in intensity from day to day, yet it could not be present each morning or each rainy day and in a few hours disappear to recur later, as do the neuralgias. Neuralgia is an old term and so is still used as synonymous with neuritis. That there may be a neuritis of almost any nerve is the common belief. Those most often mentioned are neuralgias of the face (facial neuralgia, Fothergill's neuralgia, tic douloureux), neuralgia of the back of the neck (stiff neck), of the shoulder and arm, of the chest wall (intercostal neuralgia), of the small of the back (lumbago), of the lower tip of the spine (coccydynia), of the front of the leg (causalgia, or Morton neuralgia). These conditions differ chiefly in the location of the pain.

The treatment is similar for all. If a spondylitis is to blame, we treat the arthritis by resting the affected part. In all cases we apply the Paquelin cautery, electricity (the continuous current), the ether spray, belladonna ointment, etc., to the painful areas, and, to prevent a recurrence of the attacks, we remove any infection of the tonsils, teeth, nose, gall-bladder, appendix, etc., which might possibly be the source of the arthritis.

Among direct local treatments are: the injection of 60 per cent alcohol into the nerve; the continuous application of warm dressings; the stretching or the cutting of the nerve, a therapy especially good for causalgia. Sciatica is a very difficult disease to treat, since it is so hard to remove the cause. It is very chronic and apt to recur. When it is severe, the patient may become bedridden. The doctor must first be

sure whether or not there is infection of the tonsils or teeth, or a disease of the spine or of the pelvis which presses on, or pinches, this nerve, for, if these are removed, the patient can get entirely well. Medicines are of little value. Long rest in bed, or warm baths, may cure a case. If necessary, drugs or even pure water can be injected directly into the nerve with very good effect. Or, the nerve may be exposed by operation and then pulled to stretch it somewhat—a procedure which often succeeds in relieving the pain. True sciatic neuralgia, and this form only, is relieved best by injecting novocaine (0.5 to 1 per cent) in physiological salt solution into the lumbar canal.

Herpes zoster, or "shingles," should be mentioned here since this is a trouble not primarily of the skin but an inflammation of the ganglion of a sensory nerve (Fig. 112, *b*). Of this disease there are two varieties: the essential cases (strangely enough, apparently related to chickenpox), which may develop in epidemics, and one attack of which immunizes the patient for life; and the symptomatic group, due to cerebrospinal lues, to a great variety of spinal cord diseases, and to overdoses of arsenic. In this disease the patient **feels ill for three or four days** and often has a local atrocious neuralgia along the course of one or two spinal nerves, and on one side only. Then clusters of pustules appear in the area of the neuralgia, each of which is similar to a cold sore. These pustules appear in the area of skin from which the sensory fibres of the inflamed ganglion come. Thanks to this disease, we have been able to map out the areas of skin which each sensory spinal root supplies. When a spinal ganglion of an upper thoracic spinal nerve is affected, the crop of pustules **forms a perfect band half way round the body,** from spine to midline in front, hence the name "zoster" or "zone."

Herpes zoster of the face, especially that involving the nerve to the conjunctiva, may be a very disfiguring disease. The vesicles themselves do not persist long, but they **leave a band of scars** which will endure for life. After the eruption has healed, the distressing **burning neuralgic pains** may con-

tinue even for months. The treatment of shingles is very unsatisfactory. Morphia or codeine may be necessary at first. The skin lesion, as soon as it appears, may be covered by collodion, and, if the vesicles rupture, an ointment containing 1 per cent cocaine in lanolin is advised. If syphilis is present, this should be treated intensively. Large doses of strychnia are recommended. Later, if the pain is unendurable, it may be necessary to cut the proper posterior spinal nerve root.

FIG. 120 A.—Herpes Zoster, front view. FIG. 120 B.—Rear view of Fig. 120 A.
(Indiana Univ. Dept. of Illustrations)

Peripheral neuritis is a form of neuritis which affects many nerves, as a rule the same nerves on both sides of the body, and especially those of the arms and legs. With it there is some pain in the nerves themselves, tingling in the fingers and toes, partial loss of sensation in the skin, and more or less paralysis of the muscles which the affected nerves supply. Those muscles most paralyzed are the extensors of the wrists and the flexors of the ankles, with, as

result, "wrist drop" and "foot drop." Ask the patient to hold his hands out horizontally and they hang limply down. Since he cannot lift his toes from the ground, he raises (at each step) the whole leg high enough so that the loose dangling foot cannot drag (the steppage gait). All the muscles of the body may be thus partially or wholly paralyzed, and the patient may die from inability to use the muscles of respiration. As a rule, however, the muscle weakness lasts from one to several months, and then is followed by a slow convalescence.

Multiple neuritis may occur as an acute febrile disease following exposure to cold, wet, etc. These cases have fever, headaches, malaise—that is, all the symptoms of an acute infection. Other cases **follow the infectious diseases,** such as typhoid fever, and, more often, diphtheria. The toxin of **diphtheria** almost always picks out certain nerves, especially those to the muscles of the eyes and throat. Too much **arsenic,** either as a medicine, or even arsenic in some beers, can cause it. The most common causes of multiple neuritis, however, are **lead poisoning and the steady use of alcohol. Beriberi** (page 384) is a multiple neuritis.

For months patients with a multiple neuritis have neuralgic pains and tingling in the hands and feet. Then the wrist and foot drop become evident. Very rarely, other muscles are affected. The paralyses, with loss of the deep reflexes, and the trophic disturbances, are fairly marked. Always there are sensory changes; hyperæsthesia at first, then anæsthesia. These cases practically always get well, but it takes months. Alcoholic neuritis is often accompanied by **mental symptoms,** a psychosis (Kosikow's syndrome) with delusions in which "time and place" seem utterly lost. That is, the patient is sure he was, a few minutes ago, miles away, and will tell of events of years ago as if they were yesterday.

The treatment of multiple neuritis is first to treat the cause, e.g., to eliminate all lead or alcohol. In general, the treatment depends on the nurse more than on the doctor.

In all cases the patient should rest in bed and take a general tonic treatment, including cod-liver oil. The nerves will take months to recover, since many nerve fibres must be replaced by new ones. In the meantime, the muscle fibres may become so weak and atrophied that they are almost useless when finally their nerve fibres have recovered. The secret of successful treatment is to keep the muscles in good health by daily massage, passive movements, oil rubs, electricity, etc., and then, when the nerve is well, the muscle also will be well.

The pain of multiple neuritis may be relieved by painting the limbs with menthol, and wrapping them up in cotton battings, or by very hot fomentations.

The effects on the muscles of the **degeneration of their motor nerve fibres** is well seen in **progressive spinal muscular atrophy** called also, and more correctly, **chronic anterior poliomyelitis.** In this disease there is slow degeneration of the lower motor neuron, and often, later, of the upper neuron also. The motor cells of the cord disappear, hence their fibres in the peripheral motor nerves also disappear. The result is a slow atrophy of muscles, indicated earlier by a fine twitching of their fibres. The hands are first affected. Their muscles waste away, lose their strength and dexterity, and finally contract, forming "claw hands." Later the muscles of the forearm and then of the upper arm waste and stiffen; then of the rest of the body, till, finally, we have the "living skeletons" of the dime museums. The deep reflexes are absent or diminished. It is a disease of adult life. Its cause is not known nor is any treatment of avail.

General Diseases of the Nervous System

Cerebral hæmorrhage, popularly called "apoplexy," a "stroke of paralysis," or a "shock," is usually due to the rupture of a blood vessel in the brain. The patients subject to these shocks are men with arteriosclerosis from any cause and especially those who work very hard and who drink heavily. Along the cerebral blood vessels weak spots develop, indicated often early by the **development of minute aneurisms** the size of a pin's head. The blood pressure of these patients

usually is high; even if not constantly elevated, it will, as in normal persons, rise suddenly as a result of sudden strain, violent emotion, vomiting, etc., with the logical sequence, sooner or later, of rupture of the vessel at the weak spot, and destruction of more or less of the brain tissue around it. So many cases of stroke are due to rupture of one certain artery that this vessel is named "the artery of cerebral hæmorrhage." Unfortunately, this supplies one of the most important spots in the brain, the internal capsule.

A "stroke" due to hæmorrhage (Fig. 109) may come on without warning, although usually the patient remembers later that he did have certain premonitory symptoms; numbness of one side of the body, dizziness; "thickening of the tongue" (disturbance of speech), anxiety, disturbed vision (retinal hæmorrhage), etc. With the hæmorrhage there usually is sudden and total **loss of consciousness with complete flaccidity of the arms and legs.** In other cases, the loss of consciousness is more gradual, while in still other very slight cases the paralysis develops **without loss of consciousness.** While in coma, the repirations are slow, deep, and snoring, or Cheyne-Stokes in character; the cheeks, especially that on the paralyzed side, are blown out at each expiration. One side usually is perfectly paralyzed (hemiplegia), but this, during coma, can be recognized only by raising the arms and letting both fall together. The paralyzed arm will fall a little faster, more like a "dead" limb. There may, at first, be a convulsion: the eyes are often turned to one side,—to the side of the brain in which the trouble is. (The patient "looks at his lesion.") In a few hours (from two to forty-two) the fever begins. The patient gradually regains consciousness, and then the hemiplegia becomes more apparent. The paralysis is, of course, of the **side opposite to the trouble.** It is, at first, usually a complete hemiplegia, involving one half of the face and tongue, and the arm and the leg of the same side. The muscles of the thoracic and abdominal walls escape because these seem to get nerves from both sides of the brain. When the paralysis is of the **right side,** there is

usually **aphasia also** (see page 319). The deep reflexes on the affected side are at first absent, while **Babinski's and Oppenheim's test are positive.** When one scratches the sole of the foot forcibly with a sharp metal instrument, starting at the heel and running the scratcher with one sweep along the outer side of the sole to the ball of the foot, then across to the base of the big toe, this toe, if the person is normal, will flex strongly downward toward the sole of the foot. If, however, this toe should extend forcibly upward towards the dorsum of the foot Babinski's test is said to be positive and indicates some disturbance with the pyramidal tract. If one draws the back of the thumbnail forcibly down the shin bone, the big toe will behave much as when the sole of the foot is scratched, and its movement will have the same significance (**Oppenheim's test**). The paralysis is usually much more complete at first than it is later, since many of the pyramidal fibres are only temporarily injured (by pressure e.g.) and later recover their function. For the same reason a paralysis may entirely clear up if the hæmorrhage was not quite in the internal capsule, or, if the disturbance there is only temporary and not due to an actual rupture of a vessel. **The permanent paralysis** is usually **worst** in the arm, **especially the forearm and hand.** The leg recovers somewhat and the patient usually is later able to walk; also the face often regains almost entirely its functions. The sensations may be little affected, but, if the hæmorrhage affects the fibres from the **posterior margins of the motor cortex,** the paralyzed side will be very **hypersensitive.** Gradually the affected muscles, at first too limp, become more and more rigid, especially those of the arms, and **contractures result,** leaving the elbow and wrist stiff and flexed. After a severe shock the patient always has some mental enfeeblement, and, after even the mildest cases, the patient remains **a little more emotional and irritable** than before.

The paragraphs above have spoken chiefly of the results of the destruction of the motor area and of its fibres in the pyramidal tract, but, of course, there are **other cortical areas,**

the lesions of which will give just as definitely localizing symptoms, such as **cortical blindness,** involving one half of each field of vision (page 321); **cortical deafness; abnormalities in smell; loss of skin sensations** over half the body; inability to distinguish the **form of objects;** inability to **understand written speech,** etc. All of these "speaking" areas added together, however, comprise but a small part of the cortex. The rest of the cortex is called "silent," although changes in character have been known to follow lesions of the frontal lobes.

The early diagnosis of apoplexy may be very easy or very difficult. A man is found unconscious, breathing heavily, and with all his limbs limp. The doctor, sometimes the nurse, must decide between apoplexy, uræmic coma, diabetic coma, fractured skull, morphia poisoning, epilepsy, or a simple "drunk," and must do so in a very few minutes. The uræmic patient is pale, usually has some œdema of the limbs, and always albumin in the urine; the drunken man's breath may help, although some friend, while attempting to force whisky or brandy down his throat, may have spilled some on his clothes. The drunken man, however, is never really unconscious. Pinch him or shake him and he will resist and growl his objections; the breath of the diabetic coma case has a sweet, "fruity," odor, and his urine is rich in sugar; the epileptic has a congested face; he at first had a convulsion which someone may have seen, and often has a bleeding tongue, (but so may the uræmia case); the opium poisoning case went into coma gradually, is breathing very slowly, his pupils are pin point in size, etc. But many of these points demand some previous knowledge of the case. The urine of the patient should be examined at once for albumin and sugar; the skull for a fracture; the limbs should be raised and allowed to fall simultaneously to see whether or not one limb falls faster and more limply than the other; the eyes are examined to see if they both look to one side (brain injury, hæmorrhage), etc.

Death, in a fatal case of apoplexy, usually occurs before

the third day. A fall of temperature and return of consciousness on the third or fourth days are good signs. The paralysis may clear up entirely, but if it does, it will do so within the first month; if, after that period, there is still some paralysis present, there is sure to be some permanent loss of power, at least in the hand.

Embolism and thrombosis. "Softening of the brain" (Fig. 110) seldom means true softening of the brain, but usually a failing mind; that is, a gradually developing dementia with loss of memory, of reasoning power, etc., which accompany old age. Here there really is hardening of the brain. **Real softening of the brain** does occur, but such cases are nearly always called a "stroke" and are the **result of the closure of a cerebral artery by an embolus or thrombus.** The only difference between these and a cerebral hæmorrhage is that there is no escape of blood into the surrounding brain tissue, therefore no pressure signs. Otherwise, the cases are quite similar, since both hæmorrhage and a clot end the usefulness of that artery. When, for any reason, an artery becomes closed, the brain tissue which depends on this vessel for food dies, and becomes really soft. **Emboli,** in nine tenths of the cases, **come from a heart with endocarditis** (see page 104). Unfortunately, vegetations loosened from the heart valve often travel (because it is the straightest course) via the internal carotid artery directly to the brain, and pick out the artery to the motor area of the cortex. **The onset of the paralysis** due to an embolus is sudden and in most particulars resembles a stroke of paralysis due to hæmorrhage. In **thrombosis,** however, there are more likely to be **prodromal symptoms,** the definite cause of the trouble is not apparent, coma, if present, is much briefer and is not so deep, and the development of the hemiplegia is much slower.

A thrombus may form in any diseased artery. Its results are quite similar to those of an embolus except that they develop more gradually.

If the vessel plugged by an embolus or a thrombus supplies a "silent area," there will be no localizing symptoms,

only a suddenly developing nervousness and weakness from which the patient may never entirely recover.

The treatment of cerebral hæmorrhage, thrombus, and embolism is a problem more for the nurse than the doctor. It consists at first in keeping the patient perfectly quiet in bed, and turned on one side if respiration is hindered when he is lying on his back. If there is good reason to suspect hæmorrhage, and the blood pressure is high, the patient may be bled at once, although there is doubt as to the value of this procedure. Some would open the skull in order to relieve the pressure in the cranium, but the results of this also are doubtful. An ice-bag on the head, a hot-water bottle at the feet, and a calomel or croton oil purge at once are valuable and safe in all cases. The bladder should be kept empty by catheterization if necessary. Fluids and glucose are given by rectum by the drip method if the patient cannot swallow.

After a period of about ten days following the attack, the paralyzed limbs should be massaged gently each day, and, after about a month, electrical treatments begun, the object of both being to prevent the limbs from stiffening in an unfortunate position (contractures).

Sometimes an artery on the surface of the brain ruptures and then the blood clot gathers under the meninges and presses against the brain. **These meningeal hæmorrhages** are due especially to fractures of the skull, or, as in the case of the new-born, the pressure injury. Here, again, one particular artery is most liable to rupture, and that runs over one of the most important parts of the cortex (Fig. 109).

Paralysis agitans (shaking palsy, or Parkinson's disease) is a nervous trouble the chief symptoms of which are muscular rigidity, tremor, and muscular weakness. The cause of the disease is not known, but in a "primary case" the lesion is atrophy of the cells of the corpus striatum at the base of the brain. In other so called "symptomatic cases" there are vascular or inflammatory injuries or tumor growths in this region. Parkinson's disease often follows great exhaustion, a severe wetting or exposure to cold. The mental faculties are

little impaired. First, the patient notices that his limbs are getting stiff, **a wax-like rigidity** for all movements develops, and later the tremor begins, often first of one hand and arm, then of the other, and often, finally, of the head. **This tremor is characteristic.** It is a slow turning motion (pronation-supination) of the forearm and hand, and a motion of the thumb against the fingers, as of a man "rolling a pill." If the patient gets excited, the tremor is worse; when he makes a voluntary motion, it ceases, allowing him to do the most delicate acts, such as pick up a pin, etc. At the same time the rigid limbs are become definitely weaker. His facies, station, and gait are characteristic. Since its muscles move but little, the face has so very little expression that it is spoken of as **mask-like,** a feature which can be recognized at a glance. He stands with head bent a little forward and walks as if in danger of falling on his face. Push him a little forward and he tends to go forward faster and faster (**propulsion**). Pull him back, and he tends to run backward faster and faster (**retropulsion**). The muscular rigidity, facies, and attitude, resemble closely those of some cases of encephalitis, but otherwise the conditions differ markedly.

The only treatment for Parkinson's disease is to improve the general health of the patient. The environment should be the most restful possible. Electrical baths, massage, passive movements, and exercise, not too strenuous, all have their place. Following an attack of encephalitis the patient may develop a condition which closely resembles Parkinson's disease in that the face is quite mask-like, the station on standing is stooping, and there is general muscular rigidity. The tremor, however, is not typical of Parkinson's disease but is more often volitional in character, much more like multiple sclerosis.

Epilepsy is a disease characterized by attacks of loss of consciousness, with or without convulsions. That "epilepsy" and "fits" are not synonymous the lay mind can scarcely believe, and yet some of the most dangerous epileptics have few if any true convulsions. The mildest cases also may have

no true convulsions, only attacks of momentary loss of consciousness, **petit mal,** but these have really the same significance as have the most violent fits. One scarcely notices a petit mal. The patient for a moment as though dazed stops talking, walking or whatever else he is doing, then proceeds as before. To him the attack was merely a blank moment. He does not fall nor drop anything in his hand. Other attacks of petit mal are a little more marked, the patient gives his head a jerk, or makes some automatic motion, sometimes a very violent one. Others feel dizzy and fall. These minor attacks may occur alone at first, but sooner or later the definite convulsions of "grand mal," begin, and after that the patient will have both.

The epileptic convulsion is often preceded by an **aura,** or warning, which may be a physical or mental sensation, a flash of light, a sound, etc., but which helps him little or none, since the convulsion follows so promptly that he has not time enough to save himself. Others, however, have no aura and the first they know of their attack is after it is all over. The **convulsion usually begins with a noise,** even a loud scream, and the patient "falls as if shot," making no effort whatever to protect himself, hence often is injured. The body at first is **perfectly rigid,** with jaws fixed, hands clinched, and legs extended (the tonic stage). The muscles of respiration also **are in spasm** and so the patient gets bluer and bluer. In about fifteen seconds **the convulsive movements begin,** slight at first, then more and more violent, affecting practically every muscle in the body (the clonic stage). **The tongue** is often chewed; the stools and urine often **are passed involuntarily.** After one or two minutes the convulsive movements become less violent, the body relaxes, and the patient lies in deep coma, breathing noisily. The respirations then are chiefly abdominal. For a few minutes or even hours, he cannot be aroused, then he gradually awakes. At first, on regaining consciousness, he is confused and complains of headache and malaise. Others, however, wake up quite bright. At the other extreme, are patients who, during this period, are truly

insane, and commit crimes that afterwards they do not remember. Some patients seem to have "fits of mania" without any convulsions.

The earliest convulsions occur often only at night. The patient wakes up feeling bad, or finds that he has bitten his tongue, or has voided his urine in bed, but he may not for a long time suspect the true nature of his trouble.

True epilepsy **begins usually in childhood** and continues throughout adult life. Some patients get well, but their attacks usually began after puberty. The cases which begin before ten years of age seldom get well. For a patient to have frequent attacks means that he is a severe case, and sooner or later he will show some mental deterioration. Those whose convulsions are **followed by maniacal symptoms** are very dangerous. Whether or not some intellectual persons, even some famous persons whom history records as having had fits, were real epileptics or not is a question. It is hard in such cases to **rule out lues and hysteria.** How rarely, if ever, a true epileptic is efficient is indicated by the fact that in some States epileptics are classified in the same legal group with the insane and feebleminded. While epilepsy itself is seldom inherited (that is, while epileptic parents seldom have epileptic children) yet in the production of this disease heredity is very important indeed, since epilepsy develops especially in the families of the neurotic, the insane, and the intemperate.

During a fit some object should at once be placed between the teeth of the patient to protect the tongue. He should then be stretched out on the floor, or put in a reclining posture, the clothes about the neck and chest loosened; he should then be let alone.

In the treatment of epilepsy, there are two problems to consider: the **condition itself,** and the **immediate reasons** for the convulsions. For illustration, a spark of fire and a keg of gunpowder are a dangerous combination. Either one alone may be safe. The tendency to epilepsy is the "gunpowder"; keep all "sparks" away and there need be no convulsions.

The "sparks" may be quite common and trivial conditions: eyestrain, nasal pressures, constipation, indiscretions in diet, troubles in the pelvis, etc. Control these and the number of fits will lessen, indeed the epileptic need have no more convulsions; yet, even so, he is still an epileptic.

In the general care of an epileptic **a definite program** should be mapped out, one which will cover years, and which should be followed conscientiously, no matter how well the patient may feel. Bromides and luminal are the only drugs which lessen the number of fits. Sodium bromide, well diluted in milk, is the least irritating form of bromide, and is given in large doses, and for a long time. One stops temporarily when the patient complains of drowsiness, or of gastric, or cardiac distress. It is well to omit this drug two days each week, on these days giving a laxative and flushing the colon with soda solution. **Phenobarbital,** one and one half grains each night, is, in many cases, even better than the bromides. In those cases in which the fits cease entirely, the drug should be continued for at least two years after the last fit.

The convulsions come mostly at about four o'clock in the morning. Many of them can be avoided if the patient will wake every morning before this hour, walk around the room a little to arouse himself, and then go back to sleep.

The diet of epileptics should be carefully arranged, since errors in diet often seem to be the exciting cause of a fit. The patient should eat **very little meat;** in general, a purely fruit and vegetable diet is doubtless the best. The evening meal, especially **should be light;** breakfast is best served in bed. Starvation will greatly decrease the number of convulsions, but of course it is a therapy impracticable to one who must work. Acting on this suggestion, it has recently been shown that a **ketogenic diet** (page 169) tends to decrease the number of convulsions. Such a diet should contain not over twenty grams of carbohydrates a day, and only enough fat to make the Gerhardt test for diacetic acid in the urine always strongly positive.

Gerhardt's test is made by adding to the fresh urine suf-

ficient ferric chloride solution (10 per cent) to precipitate all the phospate and still leave an excess in solution. If the urine becomes a deep Burgundy red in color, the test is almost certainly positive. Some medicines, however, may cause the urine to give the same test. For this reason, one should boil vigorously another sample of the same urine and repeat the test. If this second test is less positive than the first, or even negative, then diacetic acid certainly was present in the specimen, since it is the only substance that gives this color which is removed (broken down to acetone) by heat.

Hydrotherapy, in any form which uses cold water, is one of the best tonics of the circulation.

The care of epileptics is one of the great problems of this age. Epileptic children should receive kind but firm training, and should be taught a trade that will keep them busy. The bright children should not remain in school but should be taught at home. Present practice favors their segregation in colonies, and this is certainly, from the point of view of society, the best practice. Short daily periods of vigorous exercise are very important, especially if followed by a cold bath.

Of idiopathic epilepsy, no cause can be found in the brain; but of symptomatic epilepsy, though the fits may differ little if any from those of idiopathic epilepsy, there is a discoverable cause, such as a brain tumor, or a disease, such as chronic meningitis, etc. Reflex epilepsy is due to intestinal worms, trouble with the genital organs, a foreign body in the ear, or the nose, etc.

The fits of uræmia may exactly resemble those of epilepsy. The most common condition to exclude is hysteria, but the experienced nurse seldom makes this mistake. **In hysteria** the patient never falls "as if shot," but is careful to select a safe spot, and to fall in such a manner as to avoid injury. The movements are not truly convulsive, but rather struggling in character; or, the whole body is rigid and the person trying to restrain the patient is definitely aware of resistance to his effort. Indeed, one must beware lest he be struck or bitten. The hysterical person may scream during the attack;

the epileptic never does after the onset. The epileptic fit is over in a few minutes; the hysterical often not for hours, but it may be terminated at once, sometimes by pouring ether over the skin, or by dashing cold water over the face.

The most interesting fits of all are those of the so-called **Jacksonian epilepsy.** In these, the convulsive movements always begin in one part of the body, perhaps the finger, the wrist, or the face, and then spread in an orderly manner, always the same, to other muscles. The convulsion may be limited to one limb, so that the patient may be able to watch the convulsion during its whole course, or it may soon become general and, excepting for its manner of onset, resemble one of true epilepsy. These cases are due to some local trouble in the brain, a trouble which often can be removed by operation.

The nurse can help greatly in the diagnosis of a case with fits, since she sees the attacks oftener than does the doctor. She should note the patient's statement as to the **aura,** that is, how he knows when a fit is coming on, for, if the warning is a flash of light, a sound, an odor, a sensation in a certain part of the body, that may aid in determining in just what part of the cortex the fits begin. **The muscles which twitch first** should be noted. Perhaps the convulsion begins in one finger, or a corner of the mouth, or an eyelid, etc. This is of great importance. She should at once test the **reaction of the pupil to light,** using her flashlight. During the convulsion the difference between a case of epilepsy and one of hysteria is usually pretty apparent to the trained nurse.

Acute chorea, Sydenham's chorea, or St. Vitus's dance, is a disease of which the most prominent symptoms are frequent or constant, unconscious, irregular, purposeless muscular twitchings. Whatever the patient tries to do, he does awkwardly, or fails to do because he **cannot control his movements.** While he lies quietly, if his is a severe case, the involuntary movements of the face, limbs, and trunk will continue. In very severe cases, the movements continue even during

sleep. In addition, there is great physical weakness. One side only may be affected.

This disease occurs especially in children. It seems to be an infection, for it is so **closely related to acute rheumatism, acute tonsillitis, and acute endocarditis,** that all, probably, are due to the same germ. Some cases follow a fright, or severe school work, etc. During the week before the attack the child is **often irritable, wilful, and emotional,** in marked contrast to his usual disposition. Then begin the **awkward, jerky movements.** The child spills and drops things in his hands, and makes grimaces. The movements may affect the hands only (mild chorea), or the hands and face; in some cases, however, the entire body is affected (severe chorea). The child may be unable to feed, or to dress himself, or even to talk. He is always weak. **Slight mental symptoms** are usually present, dulness, diminished attention, depression, etc., but sometimes he is really maniacal (chorea insaniens). The case lasts from eight to ten weeks, but for about six weeks more, during the convalescence, he should remain under treatment to prevent sequelæ.

The treatment is, first of all, absolute rest in bed during the whole of the illness, and also during much of the convalescence. If possible, the child should be separated from relatives, friends, and especially from playmates; that is, from everyone except those absolutely necessary in his treatment. Good nursing consists in keeping the child quiet and amused without exciting him. Cold packs once, or twice, daily will quiet the patient. The diet should be as full as possible. Hyoscine, chloral, bromides, morphia, in severe cases even chloroform, may be necessary. Arsenic (Fowler's solution) by mouth, or, better, intravenously (arsphenamine), is the best remedy. Iron also is given for the anæmia, and salts to keep the bowels open.

It is important to prevent the child of nervous parents from acquiring this disease. It is especially important to protect him from an ambitious school teacher, and to curb his own ambitions. He should never be allowed to enter competi-

tion for a prize, etc. The child in the prechoreic, wilful stage, is often injured by correction.

The movements of **habit spasm or "tic,"** are often mistaken for chorea, although they belong more with hysteria. The child with a tic makes **frequent and regular spasmodic movements.** It may be a quick nod of the head, a grimace, a twitch of the eyelid, etc., but the movement is **always the same.** The child sometimes accompanies the movement with an oath, or some other shocking word. The treatment is to separate the child from relatives, to place him in the care of a specially trained nurse, who by occupational therapy and psychotherapy will convince the patient, without criticism or scolding or useless appeals to the "will power," that it is worth while for him to change his habits.

Chronic chorea of adults, or Huntington's chorea, is a totally different and incurable disease. It is inherited, occurring especially in certain families, its movements are slower than in the true chorea of children, in fact its movements are a series of writhing contortions. Speech is affected; there is gradually developing dementia. This form of chorea is due to definite degeneration in the brain.

Migraine is an affection with periodically recurring attacks of particularly sharp, severe headaches, definitely **limited to one side of the head.** Usually the attacks are preceded by premonitory symptoms, such as malaise, hallucinations of sight, flashes of light or colors, visions of animals, persons, etc. Some patients become partially blind; some see a bright light, or flashes of dark and light spots, or **flickering zigzag lights**; some have double vision from paralysis of the muscles of one eye. Nausea and vomiting are very common symptoms. During the attacks most patients are completely prostrated and cannot endure any noise, light, etc. Some are mentally confused, or depressed. The symptoms usually are the same in every attack.

These attacks last from one to three days. They recur frequently. They are brought on by excitement, by errors in diet, etc. These headaches certainly are more than the nor-

mal headaches, since their intracerebral features are constant. It is a definitely hereditary condition, and because it is an inheritance it would seem to be an **equivalent of epilepsy,** although some writers try to group it in the allergy class with asthma or hay fever, and explain it as a proteid intoxication.

The treatment is directed, first, to the condition itself, and secondly to the elimination of those factors which may precipitate an attack. With the latter object in view, any error of refraction, any nasal deformity, or infection should be corrected, infected teeth removed, and pelvic conditions corrected. Cocainization of the nasal ganglion, the intravenous injection of peptone with a view to eliminate anaphylactic shock, and the injection of pituitary extract in the belief that this is an endocrine disorder, all have proved an aid in therapy. Certainly phenobarbital should be tried. When the attack begins a brisk purge should be given, and the patient kept absolutely quiet in bed. Coffee sometimes diminishes the nausea. Hot and cold compresses, menthol locally applied, hot foot tubs, etc., may be tried. Of drugs, a long list is recommended, including chloroform, caffeine, cannabis indica, antipyrine, antifebrin, phenacetin, ergot, etc., but medicinal treatment gives only temporary relief.

The media of practically all the arteries of the body are **under the control of nerves** which determine the size of the vessels' lumen. These nerves are **called vasomotor nerves.** Some, the vasoconstrictors, cause the vessel to contract, that is, to become a smaller tube; others, the vasodilators, cause it to dilate. The vasoconstrictors, therefore, cause the pallor of the face due to fright; the vasodilators are responsible for blushing. Other nerve fibres control the activities of certain glandular organs; and on still a third group depend the trophic conditions of various tissues. All of these nerve systems belong to the sympathetic nervous system.

Raynaud's disease is a vasomotor disorder. In the mildest cases the person complains that exposure to cold, or an emotion, gives him "dead fingers," or "dead toes." The skin of these parts, because of a primary vasoconstriction, be-

comes white and cold, and there is a feeling of deadness or of severe pains. Then, with the reaction comes vasodilatation, and the fingers or toes become red and hot, or dark blue, or even black, and the pain intense. The whole hand may be thus affected. In cases a little severer, the fingers, the toes, or the ears, etc., become livid, swollen, stiff, and very painful. This condition is popularly called **chilblains.** In the severest cases the local cyanosis (lividity) is so intense and continuous that it leads to injury of the tissues, as a result of which the skin dies and sloughs off. Usually this condition affects only the tips of the fingers and toes, or the edge of the ears, but, in rare cases, the patient has lost whole fingers, or even the whole hand. It is important, in diagnosis, to observe that the gangrene in these cases is usually symmetrical, that is, it affects corresponding areas on both sides of the body.

The treatment of Raynaud's disease is very unsatisfactory; tonics, thyroid extract, warm applications, galvanism to the spine and limbs, brief application of the Esmark bandage to cause vasodilatation, may be tried. In severe cases, operation on the sympathetic fibres which accompany the blood vessels to the affected part has proved very successful.

Erythromelalgia, or "red neuralgia," is a condition similar to Raynaud's disease. The feet especially are affected, the hands less often and less severely. The characteristic symptoms are burning pains and congestion of both feet, which begin in the ball of the feet or the heels and advance until the whole sole is involved. The pain, though almost continuous, is worse at night and in warm weather, better when the legs are horizontal, and worse when the parts hang down. It is increased on exertion; walking and standing are most painful. Though this disease is, in large degree, functional, yet often (perhaps always) the lesions of endarteritis, neuritis, or disease of the spinal cord are present. The treatment is unsatisfactory,—elevation of the feet, rest, bandaging, cold, Faradic current therapy, baking sometimes, hydrotherapy and tonics have been recommended.

Angioneurotic œdema is a disease characterized by the sudden appearance of local œdema, causing, for example, local and transitory swellings of one eyelid, one lip, a cheek, or a hand, which suddenly may become huge. The mucous membranes of the viscera also may be affected; and, therefore, the attacks are usually accompanied by intestinal colic, pain, nausea, and sometimes by vomiting. If the swelling is of the tissue lining of the larynx, rapid death may result. The disease may be hereditary, members of even five generations may be affected. This disease is often termed allergy, under which title are grouped also asthma and hay fever, although in the great majority of cases neither by tests nor by therapy can this classification be proved. Therapy is unsatisfactory. One limits the diet greatly, and tries epinephrine and ephedrine, etc.

CHAPTER XV

Diseases of the Ductless Glands

The thyroid body (gland) (Fig. 121), which lies on the windpipe just below the larynx, can be palpated very easily especially when it rises during the act of swallowing. If easily seen, however, it is enlarged, except perhaps if the patient has a neck which is long and very thin. The thyroid body is **a ductless gland** which manufactures and administers to the blood one secretion only and that an internal one (page 51), **thyroxin,** a relatively simple substance, since it can be manufactured artificially in the laboratory. Thyroxin contains much **iodine** (9 per cent by weight), in fact most of the iodine in the entire body is there. The one important function of thyroxin, and the only function of this gland of which we have any proof, concerns itself with the use of oxygen in the tissue cells.

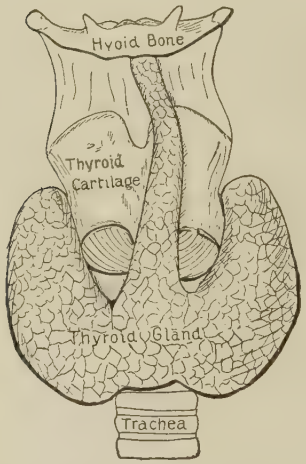


FIG. 121.—The organs of the throat. The thyroid cartilage is the so-called "Adam's apple."

Thyroxin is one of the most interesting and best understood of all internal secretions. If the thyroid gland is removed, or destroyed, **the basal metabolism of the patient falls** till the consumption of oxygen by the body is cut down almost one half, that is, till the basal metabolism is even 40 per cent below normal. The pulse rate also falls, and the temperature becomes subnormal. The patient begins to gain weight; the skin becomes dry and thick, and under it gathers a layer of mucilaginous tissue (hence the term **myxœdema**); the hair dries and falls out; the expression of the face becomes stolid, stupid and Esquimo-like; the patient becomes

irritable, suspicious, and often quite "impossible"; menstruation stops; the mind becomes duller and duller; and, finally, he may become a helpless idiot. One of the most brilliant medical attainments is the discovery that this internal secretion can be supplied by **feeding the patient daily with the thyroid gland of animals.** If a case of myxœdema is fed the thyroid gland either fresh or in the form of a dried powder (preparation spoken of as "thyroid extract"), or as thyroxin, the symptoms described above gradually disappear and, how-

FIG. 122.—Myxœdema. (This patient could only with difficulty be kept awake while photographed.)

ever distressing the condition has been, the patient in time may be entirely restored. This is **the one case of perfect relief afforded by the administration of a missing internal secretion.**

Some babies are **born with the thyroid gland absent** or practically so. In other cases the gland may, because of an early attack of scarlet fever, measles, or some other acute disease, be relatively or wholly inefficient. These children grow but little, and when they become adult may, in size and maturity, resemble a short, thick, squatty, "pot-bellied,"

anæmic-looking child of seven or eight years, but with a relatively large head, moon-like face, open fontanelles (until the fifteenth year) and with winged scapulæ, cervical kyphosis, lumbar lordosis, and narrow pelvis. The ears are large and thick, the mouth is held open, the tongue is huge, the tonsils hypertrophied, the adenoid huge, and the voice rasping. The teeth either do not appear, appear too late, or are carious and imperfect. The skin, which is cyanotic, dry, cold, and wrinkled, lies in folds. It is thick and leathery and does not pit on pressure. Pads of soft fluctuating masses of fat develop above the clavicles, in the jugular fossæ, and in the axillæ. Umbilical hernia and prolapsed rectum are common. The legs are short, the tibia are curved, and all epiphyses are enlarged when rickets is not present. The muscles are small and weak; the hands are spade-like or paw-like and cyanotic. The temperature is subnormal; the pulse is small. Most of the cases are slow, stupid and awkward, or definitely imbecile. This condition is called **infantile myxœdema or sporadic cretinism.** It can be greatly improved by feeding the child thyroid extract, and perfectly prevented if the treatment is begun early enough. Of course this therapy must be continued throughout life or the patient will relapse into the myxœdematous condition. Infantile myxœdema is not evident at birth, indeed, not until the child is weaned, since the mother will provide the infant with sufficient thyroid extract through her blood before birth and through her milk later.

The term **cretin** should be reserved for another group of children, the great majority of whom are born in "goitrous countries," whose entire prenatal development was more or less injured by the mother's inability to furnish her developing child with thyroxin. A case of infantile myxœdema may, if well treated, develop normally, but the cretin, on the other hand, **is defective when born.** He may resemble closely a case of infantile myxœdema, yet his condition is **likely to be incomplete**; for example, he may be not at all myxœdematous, although his skin is cold, dry, wrinkled, and loose, and

has a faded, yellowish color, or the subcutaneous tissue is myxœdematous in certain regions only, especially on the neck and on the back of the hands and the dorsum of the feet. His face looks senile rather than infantile. In goitre regions are found also many cases of **partial cretinism,** that is, some with endemic goitre only, some with endemic deafmutism only, others with endemic strumous imbecility. Though some true cretins have thyroid glands of normal size, over one half of them have colloid goitres. The true cretin is little if at all improved by thyroid medication.

Mild grades of hypothyroidism (diminished thyroid function) are not rare. These patients have chronic constipation; "laziness," that is, increased mental and physical inertia; a rather dry skin; dry, thin hair, etc.; all of which symptoms are much improved by thyroid extract.

A goitre is a thyroid gland a part, or all, of which is so enlarged that this organ is conspicuous. The gland may enlarge as a whole, that is, **the hypertrophy may be uniform** (simple goitre), or the hypertrophy **may not be symmetrical** and one lobe, or the isthmus, may be especially prominent. These cases may have no symptoms. Another form of hypertrophy is evidence of too great activity of the gland, and causes the profound symptoms of toxic goitre. Often, again, the goitre consists of one, or several, benign tumors called **adenomas.** Simple goitres and adenomata are common in certain regions in America, especially near the Great Lakes, and are more so in certain districts of Switzerland, Italy and Austria. Strangers, also, settling in those regions frequently develop goitres. The simple hypertrophy of the gland is to be considered **as a protective measure due to iodine starvation** and attributed to a poverty of the soil, and therefore of the drinking water, plants, etc., in iodine. It has been shown that, whereas in certain midwest cities, 40 per cent of the girls previously entered High School with hypertrophied thyroids, now, since the girls of the lower grades are required to take small amounts of iodine, not one fourth as many later develop goitre. The amount of **iodine neces-**

sary to prevent goitre is very small, only ten milligrams in organic combination three times a day for two weeks in the spring and the same amount for two weeks in the autumn. While simple goitres and most adenomas cause no symptoms, yet such thyroids are points of lessened resistance and these persons later easily become myxœdematous, or the gland so overactive that they resemble cases of Graves' disease. This activation of the gland is likely to follow sore throat, fever, emotional distress, etc.

Fig. 123.—Exophthalmic Goitre.

Hyperthyroidism, or evidences of overactivity of the thyroid gland, may be proved by the increase of the basal metabolism (from 20 per cent to 60 per cent above normal), loss of weight, an unstable heart or even tachycardia, increased psychomotor tension (that is a nervous, irritable, restless, tense frame of mind), fine tremor of the fingers, etc., symptoms which may be produced by feeding large doses of thyroid extract to a normal person.

Exophthalmic goitre, or Graves' disease, is a condition with all the symptoms of hyperthyroidism and with many others also. The thyroid is usually enlarged, seldom much; it may not be enlarged at all. This disease, however, is far more than an "oversecretion" of this gland. "Overactivity" there is in plenty, but the disturbance would seem to start back in the sympathetic nervous system. One of its results would seem to be the production by this gland of a wrong secretion (dysthyriosis).

Exophthalmic goitre gives important symptoms: bulging eyes (exophthalmos), and certain nervous disturbances in the movements of the eyes, so that, among other signs the

white of the sclera, when the eyeballs roll down, can be seen above the iris; **loss of weight**; **a high basal metabolism**; a constantly **rapid heart** (tachycardia), the pulse ranging between 120 and 160, **rapid tremor of the fingers** and hands; a **restless, apprehensive state of mind,** best described as "crystallized fear"; profuse **sweating**; profuse menstruation; and diarrhœa.

As in myxœdema, so in exophthalmic goitre, there are many cases of such slight grade that the diagnosis is difficult. These are often diagnosed as cases of nervousness, heart disease, etc. The condition of those patients **who have progressed to the last stage** of the disease is shocking. They become emaciated, intensely nervous, delirious or even insane, while the heart literally races to death.

The treatment of these cases is only fairly satisfactory. Some recover without treatment; some recover during a "rest-cure"; others, after X-ray treatment; and others, after infected tonsils have been removed. The best medical treatment is **free iodine** (e.g., Lugol's solution, ten drops, three times a day for a few days), which suggests that the trouble is really a dysthyriosis. If the case is severe, **much of the gland (subtotal thyroidectomy) should be removed.**

The parathyroid glands are four small glands about the size of a bean situated behind and embedded in the thyroid gland. These also are thought to furnish an internal secretion. When they are removed a fatal tetany results. **By tetany** is meant a general muscular tremor with spasmodic or incoördinated contractions following any effort to make a voluntary movement. In case of the most typical spasm there is tonic (steadily tense) flexion of the arms at the elbows, of the wrist, and of the fingers at the metacarpophalangeal joints. There is also great muscular irritability, a muscular spasm following pressure on its vessel or a tap on its nerve. There is also, at times, spasm of the larynx and even convulsions.

Just what these glands do we do not know, but, since tetany can be relieved by calcium administration, there is

evidence that they have some influence on **calcium metabolism.**

The internal secretion of the pancreas is called **insulin** (or iletin) a product of its islands of Langerhans. This the body cells must have in order that they may use glucose (see page 51). Insulin accelerates the oxidization and storage of glucose in the muscles and regulates or restricts the production of sugar in the liver from noncarbohydrate materials (e.g., the diabetic person tends to produce more glucose from proteid and possibly from fat than does the normal man).

The adrenals also are glands with an internal secretion only. When they are entirely removed death results in from a few hours to three days. When, for any reason, these glands are progressively destroyed the condition known as Addison's disease results. Since the cause is usually tuberculosis, we have mentioned Addison's disease under that heading (see page 457). Other diseases also, such as cancer, atrophy, inflammations, etc., may destroy these glands and produce almost the same clinical picture as tuberculosis.

An adrenal gland is really made up of two distinct organs, which exist separately in some animals, but which are represented in man, one by the cortex and the other by the medulla of these organs. It is from the medulla that the very useful drug **epinephrine or adrenalin** is obtained. This, if injected subcutaneously, makes the media of the blood vessels contract, **raises the blood pressure,** slows and strengthens the heartbeat, relieves asthma, etc. Whether epinephrine is the normal internal secretion of these glands, or a reserve secretion, or merely a useful drug which we can obtain from this tissue is an open question. It certainly is true that persons with Addison's disease have very low blood pressure.

The pituitary body is a gland about the size of a small cherry situated just behind the eyes. It consists of an anterior glandular portion and a posterior nervous portion between which is an intermediate portion of doubtful nature. The extract of the posterior lobe, that very valuable drug called

pituitrin, has a stimulating effect on plain muscle and on renal cells (note its use in diabetes insipidus, page 276), while the anterior lobe is possibly concerned with body growth and metabolism. These, however, have not yet been proved to be physiological functions of this gland. For years it has been assumed as proved that the pituitary gland was important in determining the **growth of the body.** It is known that certain disturbances of this gland, especially tumors, if they begin early in life, produce **giantism.** The individual thus affected may be over eight feet tall and huge in all proportions, yet he is so feeble that finally he cannot even stand up. If, however, this same trouble begins during adult life the **giantism affects only the tips of the body, that is, acromegaly,** and there is great enlargement of the feet, hands, superciliary ridges, molar eminences, nose, chin, etc. These tumors cause also terrible headaches, and partial blindness from pressure of the enlarged pituitary gland on the optic chiasm. Other cases with disease of the pituitary body develop a condition called dystrophia adiposo-genitalis, or **Froelich's disease,** which is characterized by obesity and marked underdevelopment of the sexual organs. In the production of these conditions perhaps the importance of the internal secretions (if any exist) of this gland has been overestimated, and the real cause is the almost inevitable disturbance by the tumor of the brain just above this gland. At least it now is well proved that this gland can be removed without death of the animal.

The thymus "gland" of children is a mass of lymphatic tissue resembling that of lymph nodes, which lies just beneath the sternum. This organ is present in all normal babies, it grows very slowly till puberity, and then slowly atrophies till only traces can be found. Its persistence or enlargement in children is supposed to explain asthma (thymic asthma). When it persists the condition is known as **status thymicolymphaticus,** in which there is also a persistence of the enlarged lymph nodes over the body, large tonsils, usually a large spleen, and a small heart and aorta.

Persons with this condition are particularly susceptible to acute infections, which run a severe course, and to emotional instability; they are particularly liable to sudden death. That the thymus is a gland has never been proved; that it is responsible for any of the conditions attributed to it, is doubtful; its importance may be rather that its presence calls attention to serious (and as yet not understood) conditions associated with the failure in involution of these lymphatic structures, which should disappear as the body matures.

One rare condition which may here be mentioned, since there is some evidence, although as yet not sufficient, that it belongs in this group, is **Dercum's disease,** or **adiposis dolorosa,** a disease characterized by a great increase of fat, which is painful to the touch, and which is usually deposited unequally over the body. The mental condition of these patients is somewhat subnormal. A few cases have improved when fed with thyroid extract but more have not.

CHAPTER XVI

Constitutional Diseases

Gout is a disturbance of metabolism because of which uric acid, as sodium urate crystals, is deposited about the inflamed joints, especially the great toe, and as little masses called **tophi** in the ears and on the knuckles.

Gout is often described as the result of too much food and too little physical work, but it is better described as the result,

FIG. 124.—Tophi of gout.

primarily, of a certain form of chronic nephritis, the distinguishing feature of which is the **inability of the kidneys to eliminate uric acid well.** The amount of the acid in the urine, therefore, is always abnormally low while it accumulates in the blood and is deposited in the cartilages at those points where the circulation is poorest, that is, in the cartilage of the ears, fingers and toes, where tophi form. The disease has

been ascribed to the habitual consumption of too much meat, to fermented alcoholic drinks, and to the use of lead (in the case of painters, etc), but the worst forms would seem to be inherited.

An attack of gout usually begins in the early morning, with agonizing pain in the first joint of the big toe, and later in the other joints of the foot. The joints become swollen, red, hot, and exquisitely sensitive; the patient feels as if the foot were in a vise. There is fever. Later in the morning the pain abates, may even disappear, and the patient can walk about with joints which still look much inflamed. The next night the pain returns, and so on for from five to eight days, its severity gradually diminishing each time. These attacks usually recur, often with intervals of some months. Almost any joint may be involved, but the great toe rarely escapes.

In addition to joint troubles, there are many other symptoms which possibly are **evidences of gout** and which are most common in those who inherit this condition. Among these are: gastrointestinal attacks, with pain, vomiting, diarrhœa, and depression; skin diseases, especially eczema; dyspepsia; arteriosclerosis and all its resulting troubles, etc. All of these, it will be noted, might be the results of chronic nephritis and arteriosclerosis.

The only sure test of gout is the **presence of tophi in the skin,** from which one may with a needle obtain the typical crystals of sodium urate. In the absence of these, the presence of gout is indicated when the uric acid in the blood rises to 5 mg. per 100 c.c.s. or over, provided at the same time the urea and creatinin of the plasma are not increased, in which case one would suspect uræmia.

As a result of chronic gout the joints become considerably deformed. The deposits of uric acid sometimes form on the knuckles knobs which may ulcerate through the skin (chalk stones).

The gouty patient should be a total abstainer from alcohol, temperate as concerns food, and regular in his exercise in the open air. He should keep his skin in good condition.

The diet should be free of purin bodies, that is, kidney, liver, sweetbreads, brains; and rich in milk, eggs and fruits, with water in great excess. Diets should, on the whole, be avoided, since they are usually extreme in some point or other. It is better to begin treatment by starving for a few days and then begin with a very limited milk diet, adding next milk and eggs, and later a mixed diet of limited quantity. Between attacks, cinchophen (atophan), 0.5 grms. each two hours, six doses a day (that is, 3 gms. per day) is prescribed for each third day.

During the **acute attack** the patient should be purged, a diet of milk only allowed, the foot wrapped in hot compresses and elevated, and wine of colchicum given to relieve the pain and inflammation.

Diabetes mellitus is a disturbance of metabolism. Glucose, the very important fuel of the body, is not well used; it therefore accumulates in the blood, and is excreted by the kidneys. Although nephritis may coexist, yet diabetes mellitus is not a disease of the kidneys, for their duty is to remove from the blood all glucose in excess of about 0.2 per cent. Nor is it a disease of the blood. The truth is that the tiny cells, the little living workshops of the body, cannot use glucose well; yet they are not diseased. The trouble is chiefly in the pancreas. The pancreas furnishes the blood with an internal secretion, recently isolated by Banting, called **insulin,** which must be present in order that a cell may use its glucose.

The importance of insulin is evident from the fact that it makes not only the combustion of glucose possible, but this combustion of glucose, in turn, makes that also of the fat and proteid possible. **Fat,** which furnishes the body with most of its heat and energy, produces, when it is split, ten per cent of its weight in glucose and ninety per cent in fatty acids, and these fatty acids burn well only in a "glucose fire." When not enough glucose is burned to consume these fatty acids (one gram of glucose will burn more than one and a half grams of them) the products of their incomplete combustion will accumulate in the tissues and are definitely

poisonous to the body. For illustration, suppose you desire to burn up a heap of rubbish. You would find that it burns slowly producing much smoke; but if you should mix with it kerosene oil or dry shavings, the whole would burn well. So, when our tissue cells have difficulty in oxidizing fatty acids, they produce, while doing so, products of incomplete combustion some of which are distinctly poisonous. This explains the increased number of cases of diabetic coma when the carbohydrate-free diet was popular. It should be a general rule of diet in diabetes that the patient may have only as much protein and fat as can be completely oxidized by the amount of glucose which he can successfully burn. If a normal person is put on a strictly carbohydrate-free diet there will within a week appear in the urine the so-called **ketone bodies**; acetone, diacetic acid, and B-oxybutric acid, an acid similar to that which gives the odor to rancid butter. These acids are present in large quantities in the urine of severe diabetics who are not carefully dieted, and may finally cause coma and death, perhaps by intoxication—"acid intoxication,"—perhaps by using up the alkali of the body and thus causing an "alkali starvation."

The proteids, in turn, when they are split in metabolism, produce fifty-eight per cent of their weight as glucose and forty-six per cent as fatty acids. Consequently, the general problem of metabolism may be stated simply as follows: carbohydrates appear in metabolism as glucose; fats, as glucose and fatty acids; and proteids, as amido-acids, glucose, and fatty acids; it is insulin that makes it possible for glucose to burn, and the oxidizing glucose aids the fatty acids to burn.

The essential element in diabetes mellitus is not the glycosuria but the **reduction of the patient's ability to use glucose.** Theoretically, therefore, a case of diabetes mellitus might live a long life and never have glycosuria—provided he never taxed his glucose-burning power to the level of his limitations, just as a person with a slight heart lesion might never have "heart disease," provided he never overtaxed his

injured heart. Both patients might never have reason to believe themselves sick if only they always lived "within their income," the diabetic, within the limits of his pancreas' ability to furnish enough insulin to answer the needs of each day.

To find **glucose in the urine does not necessarily mean diabetes.** We must find it during long periods of time. It is often present for a few days after injury to the head as well as in other conditions.

A normal person can ingest and warehouse (store up in the liver and muscles) **an indefinite amount of starch** without causing even the slightest glycosuria. The reason for this is, probably, that the long time required to digest and absorb this starch allows the body sufficient time to warehouse the glucose which it furnishes. Each normal person has, however, a very definitely limited ability to warehouse pure glucose if ingested as such. If he were to eat at one time 300 Gm. of sugar (glucose or even cane sugar) he would quite certainly excrete some of it in the urine, for the normal body simply cannot use or store up that large amount on such short notice. In other words he would have overstepped his **assimilation limit,** which is determined by giving him on successive days larger and larger amounts of glucose until we reach the point at which sugar does appear in the urine. But, if a person loses sugar in the urine after a 100 Gm. dose, then diabetes mellitus may be suspected, for the normal person has a higher assimilation limit, or to use another technical term, a greater **tolerance to glucose,** than that. The assimilation limit of a very mild diabetic patient is only a little lower than normal, while the severe cases tolerate very little sugar. While the normal man has practically no assimilation limit for starches (bread, etc.), yet the diabetic has a very definite limit, and what, in each case, this is, we should know if we are to diet him correctly. It is also important to determine **the amount of a patient's blood-sugar;** that is, the number of milligrams of glucose in a hundred cubic centimeters of blood, both during a fasting period (e.g. before breakfast)

and after a meal containing a known amount of glucose, since this aids us to determine the severity of the case, also the dangers of an overdose of insulin in a patient on that treatment.

If glycosuria were a disease, its treatment would seem fairly simple, but the facts are quite otherwise. **Glycosuria is a symptom,** not of one, but of several conditions. Just what the underlying disease is, is hard to say. First, while in the young patients no lesions are found, yet in many adults at least, there are several diseases which cause glycosuria because they happen to attack the pancreas (lues, cancer, hæmatochromatosis); second, the resulting disturbance of the pancreas is likely to affect its external secretion also; third, glycosuria itself tends to injure the body; fourth, the ketone bodies which accumulate in the blood also produce injuries; and, fifth, that disease which happens to attack the pancreas may injure other organs also and cause many complications and sequelæ which require still other explanations. All of these separate disease processes require individual attention.

Some diabetics have inherited their disease; some date it back to a severe mental strain, nervous shock, injury, or to an acute infectious disease; in still other cases no reason can be assigned. The anatomical cause is described on page 255.

In the following pages, the term "sugar-free," when used of the urine, means that no sugar is present, and "glycosuria" that it is present. Practically the only sugar excreted in diabetes mellitus is glucose or grape-sugar. When speaking of diets, also, the term "carbohydrate" is used to include all sugars and starches of the foods. We eat very little glucose as such.

The onset of diabetes mellitus is often gradual, the first symptom, usually, an increase in the amount of urine passed (polyuria). With this polyuria there is intense thirst (polydipsia) since much water is needed to excrete this sugar. When the amount of sugar excreted is diminished by dieting, the amount of urine at once diminishes, and the thirst lessens. It used to be said that diabetics had voracious appetites; that they had a terrible craving especially for sweets

and starches; that their conscience is affected, for to get these they would use all kinds of deceit; that sometimes they would go on "carbohydrate sprees" and gorge themselves with ice cream, cake, and candy. But now we know that all this was our own fault, for we were feeding them far too much fat and proteid in proportion to the carbohydrates of their meals, and as a result their tissues were overfilled with the products of partly burned fatty acids, which made them crave madly any food that would furnish glucose.

Unless his glycosuria is checked and the diabetic patient able to remain sugar-free while using sufficient food it is the tendency of diabetes mellitus that emaciation begin, and the case go steadily down hill. The lean and hungry diabetics are usually very severe cases; the younger the patient, the more severe the disease. The "fat diabetics," whose glycosuria as a rule begins after middle life, may run a long, mild course, some almost without symptoms. It is a general rule (and a very important one) that even **one sugar-free day so increases the patient's tolerance** to carbohydrates that one may begin safely, the following day, to allow an amount of carbohydrates which the day before certainly would have produced a glycosuria; and that this amount of carbohydrate can be increased slowly day by day even to surprising limits, without sugar appearing in the urine, provided we do not at any time overstep the patient's slowly increasing tolerance. It is a general rule, also, that if even a slight glycosuria is constantly present, the tolerance to carbohydrates tends progressively to decrease.

The urine in mild cases may be normal in amount, but usually it varies from 3 to 4 litres per day; in severe cases, an output of from 4 to 10 litres is not rare; and in the severest and badly treated cases it may reach 20 litres or more each day. **Its color** is a pale greenish yellow, and its specific gravity is high, from 1025 to 1045 or over. The **amount of sugar** varies with the diet. If the patient eats all the carbohydrates he wants it may reach a pound or more

a day. If on a rigid diet, however, the otherwise untreated case seldom loses more than three ounces a day.

The nursing care in diabetes mellitus is, for the ambulatory cases, entirely educational; but for those with serious complications or threatened coma, it is most active. The patient, while in the clinic, should learn how to plan and calculate his diet, how to examine his urine, how to meet many problems of personal hygiene, especially the care of the skin and of the feet, and should gain such clear understanding of his problem that he can continue his treatment while at home.

A case of diabetes mellitus may be said to be **mild** when the patient is made sugar-free by merely omitting a few of the commoner carbohydrates without greatly reducing his total diet. It is more severe, yet still moderate, when the patient is, and remains, sugar-free on a carefully balanced and weighed diet, yet one sufficient in amount to allow him to work comfortably. A preliminary period with insulin may be necessary to make this possible. **A case is severe** if to keep the patient sugar-free and yet strong enough to work with comfort requires not only careful dieting but the daily administration of insulin.

Formerly, we fed patients with diabetes mellitus a **diet of proteid and fat** and as free as possible of carbohydrates. These cases did very badly since this diet greatly favored the production of coma. Then Joslin recommended a diet based on Allen's experimental work. The patient was first starved until his urine contained no sugar. This might take from one to ten days, usually three or four. During this time he was given all the water he wanted, coffee, bouillon, etc., but no food. After the urine had become sugar-free the patient was fed an increasing amount of carbohydrates, to which later protein and, still later, fat, were added, and these increased daily until sugar appeared in the urine. The diet then prescribed was about one half that which produced the glycosuria. Then Woodyatt, following Neuburgh and Marsh's work, improved this diet much **by adding more fat.** He taught us to calculate not so much what the patient ate, but

rather the amount of glucose and fatty acids which such a diet would produce in the tissues. This is the basis of our modern diet. Following it we now treat the mild and moderately severe cases with success, but before the days of insulin the severer cases were continuously half starved and so were weak, unable to work, hungry, and unhappy.

Now * **with insulin, we may at the first visit prescribe for the patient his final diet.** We calculate the food necessary to cover his basal metabolism, increase this by 50 per cent in order to cover his needs while at work, and keep him sugar-free by means of insulin. Such a diet would contain about 1 gm. of proteid per one kilo of body weight (the adult on full diet needs at least 0.75 gms., the young adult at least 1 gm., and the child about 1.5 gms. of proteid per 1 kilo of body weight), and enough carbohydrate and fatty acids (in the proportion of from 1:1.2 to 1:1.5) to furnish the necessary total food value. This diet is divided into three meals, not necessarily equal. The urine secreted after each meal is collected separately and the sugar in each determined. For illustration, if the meals are to be served at 7:30 A. M., 12:30 P. M. and 6.00 P. M., then the urine is collected from 7:15 A. M. to 12:15 P. M., from 12:15 P. M. to 5:45 P. M., and from 5:45 to 7:15 A. M. In very severe diabetics we may also collect separately the night urine, from 9:30 P. M. till 7: 15 A. M., since the inactivity of sleep favors glycosuria. Knowing how much sugar is likely to follow each meal, we then, fifteen minutes before each, inject the proper amount of insulin to make that period sugar-free. Then, since even one sugar-free day improves the patient's tolerance, we at once begin to reduce the doses of insulin, beginning with the noon dose, which can soonest be dispensed with, until if possible no insulin is given and yet the patient remains sugar-free. Later we examine but two specimens a day, from 7:15 A. M. till 6:15 P. M., and from 6:15 P. M. till 7:15 A. M., and later we examine only one, the mixed

* The details of diets should be gained from special text-books.

twenty-four-hour specimen. Soon, only an occasional specimen need be examined.

In planning the diet the doctor prescribes its total caloric value and the percentage relations of its foodstuffs, but the nurse must select the individual foods, see that they are properly prepared and in the most attractive, digestible, and acceptable form possible. She should see that the restrictions and limitations of the diet cause the patient as little distress as possible; that the meals resemble as nearly as possible the diet to which that person was accustomed when well, and that his tastes are considered as far as the limitations permit; that he eats slowly and chews the food thoroughly. This means that she should understand, as far as is possible, his racial customs, his family habits and prejudices, and his personal likes and dislikes. Any system of diet is doomed to failure if the same general formulæ are used for all the diabetic patients on any one ward, regardless of their race and family habits.

The nurse should watch for and report all **symptoms suggesting coma,** a complication which usually can be prevented if treated early. It always threatens the severe cases and may be precipitated by various factors: ether anæthesia, impaired function of the kidneys, constipation, mental excitement, fatigue, infections, exposure and chilling, overeating, fasting, sudden changes in the diet, improper regulation of the diet, such as sudden increase in the fats when the carbohydrates are low. It is surprising how unexpectedly it may develop, but also how early the watchful nurse can detect it if she watches for loss of appetite, nausea, vomiting, headache, listlessness, drowsiness, weakness, vertigo, ringing in the ears, disturbances of vision, excitement, or delirium. Later, begins the typical diabetic dyspnœa or diabetic air-hunger, that is, the very deep and straining respiratory movements, although there is no impediment to respiration. The breath has a sweetish odor. Soon the patient becomes drowsy, and, later, comatose. The blood at such times will show a high blood sugar, an increased percentage of fat, and

a reduced power of the plasma to hold carbon oxide. The urine will then show by simple tests the presence of diacetic acid and acetone, both of which are derived from B-oxybutyric acid, which also is present, but which is much harder to demonstrate. Even then, after coma has begun, if treatment is at once begun, the patient soon will regain consciousness. **A patient with oncoming coma** should at once be given intravenously thirty grams of glucose and twenty units of insulin. This should be repeated each three hours. The result is a glucose flame in which the ketone bodies will be oxidized. He is put in bed, kept warm with blankets, hot water bottles, etc., and given an excess of water at regular intervals, at least two quarts each four hours. If the patient cannot take this by mouth it is given by rectum or intravenously. Some doctors give bicarbonate of soda by mouth, or rectum, or intravenously, to neutralize the acidity of the blood, but with insulin this is unnecessary.

To quench the thirst of diabetic patients with polydipsia, water, tea, coffee, and clear meat broths properly seasoned are the usual fluids allowed, and are given hot to avoid the loss of heat necessary to warm a cold drink. (It must be remembered also that the large amount of **urine voided** at body temperature **robs the body of heat).** Salt should not be restricted.

Plenty of sleep at night is essential in all cases of diabetes while in the hospital. The patients should not, however, be kept in bed during the day (unless some complications require this) since **exercise or light work is always desirable,** the amount depending on the condition of the patient and the total caloric value of his diet. Outdoor exercise and those which are enjoyable and diverting give the best results. The amount of exercise the patient takes may be increased by training. Muscular exercise keeps the muscles in good condition and improves the patient's mental attitude, but, what is more important, it increases his carbohydrate tolerance and allows him a more liberal diet and a much greater variety of food. It also makes the transition between

the periods in the hospital and the home and later when at work less abrupt and more natural. **Over-exertion and fatigue** are to be avoided, since fatigue lowers the assimilation limit and raises the blood sugar, and, in severe cases, may predispose to coma. In cases on insulin treatment, also, excessive amounts of exercise may, by aiding in the combustion of sugar, lead to a hypoglycæmic reaction. The patients should also avoid exposure to cold; cold water or ice cream (even though made in accordance with the diet allowed) should not be given because heat is necessary to warm them. **Conservation of energy** is particularly important in old people. For this reason, for them fasting is usually avoided.

The emotional life of the patient would seem to be important in determining the course of diabetes. The onset of this disease often dates from a severe mental strain or nervous shock. All through its course worry, insomnia, and all depressing emotions lower the patient's carbohydrate tolerance. A carefully estimated diet and insulin dosage may be rendered unsuitable because of one sleepless night, one day of disappointment, etc. In severer cases any nervous strain, as worry or anger, predisposes to coma. On the other hand, a nurse often can interpret a complaining frame of mind as a symptom of the disease, and a more cheerful attitude as a sign of improvement. Formerly, prolongation of life meant for the severe diabetic the unhappy, hungry, weak, idle life of the semi-invalid. Now, thanks to insulin, a diabetic patient, except while in the clinic and during periods of prescribed starvation, may feel well and strong enough to do a full day's work without unusual fatigue. Nevertheless, the need of watching each meal so carefully and the frequent use of the hypodermic needle make it hard to be patient. The fact that their disease is not curable is naturally depressing, yet the patients actually do keep much happier than one would expect.

Among the complications of diabetes are skin infections (page 379), severe itching (pruritus), gangrene of the fingers and toes, or even of a whole limb, neuralgias, paralyses, cata-

ract of the eye, and, as **terminal infections,** pneumonia, gangrene of the lung, pulmonary tuberculosis, or blood-poisoning. Thanks to insulin, these terminal infections are not so much dreaded as formerly.

Arteriosclerosis is usually present in the diabetes of adults, particularly in the severe forms, and greatly adds to its dangers.

It is extremely important in diabetes mellitus **to keep the skin active,** clean, warm, free from irritation, from infections, and generally in good condition. Partly because of the loss of water in the urine, more because of the hyperglycæmia, and still more because of the atrophic changes due to the arteriosclerosis and spinal cord changes common in this disease, the skin is apt to be dry and harsh, the seat of intolerable itching and eczema, and very susceptible to infections, such as boils, carbuncles, bed-sores, and gangrene. The urine is irritating and may cause pruritus. Emaciation adds to the danger of **bed-sores. The care of the feet and toes** is especially important since infection and gangrene of the toes are among the largest indirect causes of death. Any break in the skin heals with great difficulty. The body must, therefore, be kept scrupulously clean by daily baths, and stimulated by warmth, exercise, and massage. Gangrene may be prevented by avoiding conditions which lead to arteriosclerosis, and by improving the circulation of the extremities.

Care of the mouth and teeth is most important. The mouth is usually dry, the tongue dry, red, and glazed, the gums swollen, and stomatitis common. The teeth are often in poor condition, and their infections increase the severity of the diabetes. All symptoms suggesting a complication should be reported immediately. These not only are serious in themselves but they lower the carbohydrate tolerance, and interfere with the treatment. Since a diabetic patient, particularly when on a low diet, should never be exposed to infection, a nurse with a cold should not be assigned to him. Constipation should be avoided. The patient should be warned, however, that a "slight" infection such as a cold in

the head, a boil on the skin, or a day of worry, a sleepless night, or great over-exertion, makes it necessary that he be "checked up" again, since it changes his sugar tolerance considerably; that is, he becomes a "new" case.

A patient on insulin treatment should be warned to **carry with him some syrup or milk chocolate,** since at any time, but especially late in the afternoon, the dose of insulin may reduce the blood-sugar so much that a hypoglycæmia (below 70 mg. per 100 c.c.s. of blood) develops, whereupon the patient feels extreme hunger, a sudden nervousness, a sweating, trembling, internal tremor, or weakness, and becomes hysterical or even unconscious. If he takes at once syrup or other form of sugar, in a few minutes he will recover. Sometimes the attack, a convulsion for illustration, comes on so suddenly that the patient cannot help himself. This condition often develops during trips away from home when the diet is disturbed, a meal is omitted, or part of a meal is lost by vomiting, or after the patient takes unusual exercise, which consumes so much sugar that the insulin given proves to be in excess.

The prognosis in diabetes has always been considered bad, and still is, especially if the patients are young. By substituting the lower balanced diets for the older carbohydrate-poor diets we saved many diabetics from death by preventable and unnecessary coma. Whether insulin will increase the expectancy of life for the diabetic is still a question. It does, however, render the patient almost normally comfortable and efficient in his business during those years which formerly were spent as an invalid.

Diabetes insipidus has been described on page 275 since polyuria is its one essential symptom.

CHAPTER XVII

Deficiency Diseases

Rickets, a disease of infants especially, is characterized by alterations in the bones at those points (the epiphyses especially) where and at the age when they are growing most actively, and by emaciation of the entire body. It comes on insidiously, most often between the sixth and eighteenth months of life. There is fever, the child becomes irritable and restless, especially at night when he is always throwing off the bedclothes. He screams if touched, as if his whole body were sore. He sweats profusely, especially the head, so that the pillow is soaked. There are also digestive disturbances. The child will **not learn to walk till late,** and, if he has already learned, the rickets will for a time put a stop to his walking. The mother fears he is paralyzed. There is great weakness of the muscles. The child may lose weight rapidly, or he may stay plump but flabby. **The ends of the ribs and of the long bones swell** because of an overgrowth of soft bone. **The child's head** is large, and square in shape. The skull often feels thin, like parchment, and the fontanelles do not close as early as is normal. The face is small, its forehead prominent, and, as a rule, **teething is delayed. The chest becomes somewhat deformed** (often "pigeon breast" is due to rickets). The "rickety rosary" of the ribs and the too large wrists and ankles persist through life. The bones of the arms become

Fig. 125.—Saber shaped shins due to rickets.

crooked if the child uses them much in creeping, and the legs bend if he walks much. These may become bowed, or knock-kneed, but the most characteristic change is a **forward arching of the tibias,** hence the name "saber shins." **The abdomen is swollen,** (pot belly), partly because the liver and spleen are big, but more because the bowels are flatulent with gas. The child is usually peevish and irritable, sleeps poorly, has convulsions and spasms of the throat, and is very susceptible to acute diseases. All grades of the disease occur, from the mildest, which are not noticed by the mother, to the severest, which produce misshapen dwarfs.

Rickets usually begins in winter and reaches its height in March. It is met with by far most often in cities and large industrial centres, and especially among negroes and immigrants. Nevertheless, it is not entirely a disease of the poor, since the most expensive diet may be faulty. It is one of the deficiency diseases mentioned on page 170 and due in part at least (although not entirely) to a **lack in the diet of vitamine D.**

In treatment there are two efficient and almost specific therapeutic agents for rickets; **cod-liver oil and ultraviolet rays.** The child should have a healthy wet nurse, or be fed on properly diluted cow's milk. He should have cod-liver oil and orange juice daily. He should be kept perfectly quiet to prevent deformities; should, as much as possible, lie, warmly clad, in the fresh air and sunlight,—sunlight, however, which does not come through window glass since this removes all of its ultraviolet rays. The ultraviolet ray treatments are of great value. Warm baths and sweet-oil rubs will greatly relieve the sensitiveness of the limbs.

Scurvy is a deficiency disease which develops **when vitamine C is lacking** in the food. It is due, especially in adults, to a lack of fresh vegetable food and in children to the use of boiled or condensed milk or proprietary infant foods. It was common enough among sailors until the use of well-preserved fruits came into common use. Among infants it is by no means, as is commonly assumed, a disease of the

poor, for it occurs in children of wealthy families, especially those between the fourth and twelfth months of life, who are reared on most expensive foods. Its chief symptom is **bleeding in various parts of the body,** into the joints, under the periosteum of bones, or from the spongy gums of the mouth, or from the bowel or the stomach. **The adult** becomes **sallow, weak, and short of breath** and soon complains of rheu-

FIG. 126.—Pellagra.

matic pains in the legs. Then the **gums bleed and subcutaneous hæmorrhages** appear over the body. Later symptoms are necrosis of the jaw which loosens the teeth, bloody diarrhœa, hæmorrhage into the muscles, œdema, and death. **A child** with scurvy is pale, restless, sore when handled, and then develops localized swellings of the limbs which are due to hæmorrhages under the periosteum of the bones, or under

the skin. This child should be given **fresh fruit juice** at once, either orange or lemon, and should be put on fresh cow's milk. The cure is astonishingly rapid, all traces of the disease disappearing often in less than a week.

Pellagra (rough skin) is a deficiency disease due to some error in the proteid content of the diet; exactly what, is not known. Its manifestations recur periodically; each annual attack begins usually in the spring, and leaves the patient worse. The disease begins with gastrointestinal disturbances, **the mucous membrane of the mouth and tongue becomes raw and ulcerated,** there is nausea and vomiting, and diarrhœa.

The skin lesions appear early. The backs of both hands, from the wrist to the last finger joint, become "sunburned," then the skin here "peels" and becomes more and more pigmented. It becomes progressively dryer, thicker and rougher, but finally thin and atrophic. After several such attacks the nervous symptoms begin, which in time end in dementia. Some patients have many such attacks, some recover, others progress to death.

The diet should be rich in meat, milk, eggs, and vegetables, especially beans. Arsenic in any form is valuable but especially as Fowler's solution. Transfusion is a great help.

Beri-beri, or endemic multiple neuritis, is a deficiency disease due to the lack of certain vitamines, **especially the water soluble B,** and is characterized by multiple neuritis, dropsy and muscular atrophy.

It usually begins with paræsthesias, pains, weakness, and œdema of the legs. In some cases this œdema, extending over the whole body, is the predominant feature (the "wet" form). In others the muscular atrophy predominates (the "dry" form). In both, rapidly developing muscular weakness is an important symptom.

The treatment is a diet rich in vitamine B, rest, purgation, cardiac stimulants and physical therapies for the atrophied muscles.

CHAPTER XVIII

The Infectious Diseases

The Pathogenic Micro-Organisms

Man is composed of many, many, cells visible to us through the microscope. In other words, man is a metazoon, as are all of the animals and plants we see about us. If now we again look through the microscope not at ourselves but around us, a new world is revealed. Myriads of tiny

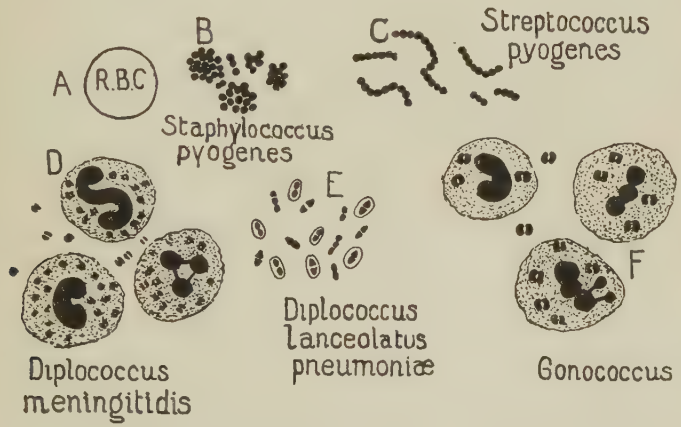


Fig. 127.—Vegetable parasites: Bacteria; Cocci. (All drawn to same scale, magnified 1000 times.) *A*—A red blood-corpuscle, drawn to the same scale for comparison of size. *B*—Staphylococcus pyogenes (aureus or albus). The cause of boils, pimples, abscesses, etc. *C*—Streptococcus pyogenes. The cause of erysipelas and "blood poisoning." *D*—Diplococcus meningitidis, or the "menigococcus." The cause of epidemic cerebrospinal meningitis. *E*—Diplococcus lanceolatus. The cause of pneumonia. *F*—The Gonococcus.

creatures, animals and plants, all living and active, swarm where they had not been suspected. Some of these tiny creatures, like ourselves, are metazoa, although much simpler than we, while others are unicellular, that it, they consist of but a single cell. **These are the so-called micro-organisms.** Some of these belong to the animal kingdom, and are called **protozoa,** others to the vegetable kingdom, and are called **protophytes,** while the most, which seem to be neither animal

or vegetable, are classed in a kingdom of their own and are called **bacteria.** Of these bacteria the tiny round ones are called **cocci** (See Fig. 127). Not all cocci are perfectly round;

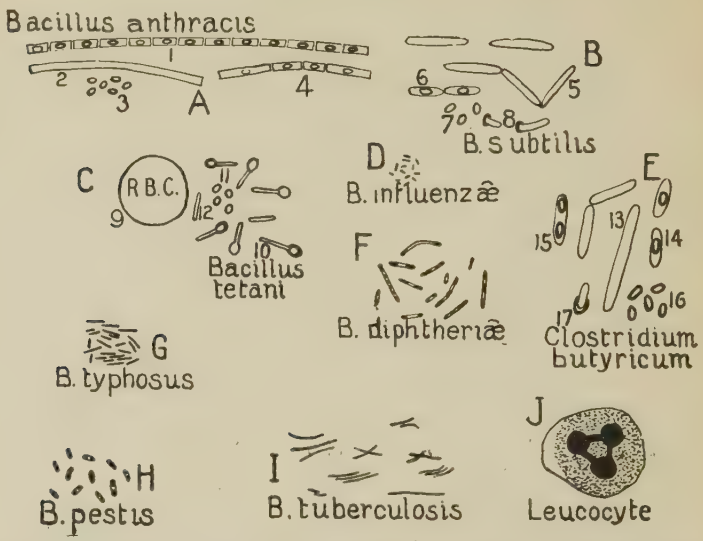


FIG. 128.—Vegetable parasites: Bacteria; Bacilli. Important disease-producing and harmless bacilli. (All drawn to same scale, magnified 1000 times.) *A*—Bacillus anthracis. A very dangerous bacillus, the cause of "anthrax," "malignant pustule," "wool-sorter's disease," etc. 1, a chain of bacilli, each containing a spore; 2, one long bacillus; 3, free spores; 4, a short chain. *B*—Bacillus subtilis. A harmless ubiquitous bacillus, also called "hay bacillus." 5, a short chain of bacilli without spores; 6, two bacilli containing spores; 7, free spores; 8, two spores "sprouting," that is, developing into bacilli. Note that this bacilli "sprouts" from the side of the spore. *C*—Bacillus tetani, or the bacillus which causes "lockjaw." 9, a red blood-corpuscle drawn to the same scale, introduced for comparison of sizes; 10, the "drumstick-shaped" bacilli containing spores; 11, free spores; 12, a bacillus without a spore on the left; *D*—Bacillus influenzæ, the cause of "la grippe." This germ produces no spores. *E*—Clostridium butyricum. A large harmless bacillus. 13, bacilli without spores; 14, bacilli containing spores; 15, a bacillus containing two spores; 16, free spores; 17, a spore "sprouting." Note that this bacilli develops from the end of the spore. *F*—Bacillus diphtheriæ. There are no spores formed by this germ. The dots are not spores, but indicate irregularity in staining ("beading"). *G*—Bacillus typhosus, the cause of typhoid fever. The same picture will do for Bacillus coli communis, Bacillus dysenteriæ, etc. No spores are produced. *H*—Bacillus pestis, the cause of bubonic plague. The dots do not indicate spores. *I*—Bacillus tuberculosis, the cause of consumption. No spores are produced. *J*—A leucocyte drawn to the same scale for purpose of comparison of size.

some are flattened together in pairs—"diplococci"; some are a little oval, or lance-shaped, as Diplococcus lanceolatus (Micrococcus pneumoniæ); some are always in groups—the staphylococci; and some always in chains, the streptococci. Those bacteria that look like a stick are called **bacilli** (Fig.

128); those that are curved, **spirilla** (Fig. 129); and still others are long threads which branch, **streptothrica**, etc.

Though the activity of these tiny creatures is ever manifest, they themselves cannot be seen by the unaided eye unless vast numbers are crowded together in colonies, seen, for example, as dots and streaks on a crust of bread. To study the separate individuals we must so magnify them that were

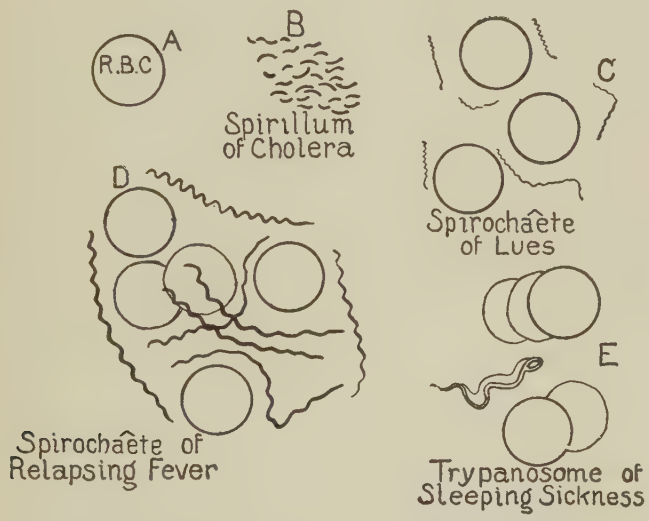


FIG. 129.—Pathogenic organisms. *A*, a red blood-cell for comparison of size. *B*, the organism of Asiatic cholera. *C*, the organism of lues or syphilis. *D*, the organism of relapsing fever. *E*, the organism of sleeping sickness.

a man magnified as much he would appear to be over a mile tall. Escape from this world of the micro-organisms we cannot, nor should we try, for the life of the world, including our own life, is dependent on them. In the great chain of existence, the carnivorous animals use as their food the herbivora, and the herbivora, plants. Plants, in turn, feed on the simplest gases—carbon dioxide, ammonia, salts, and water, and these, in part at least, arise only from the decomposing tissue of dead plants and animals. Suppose that the putrefying bacteria should suddenly cease their activity. Then each

plant and animal when it dies would be embalmed by the sun, there would be less and less food for plants, and so finally they would die, then the herbivorous animals and finally the carnivora, all would die, if this one link of micro-organisms could be dropped from the chain. But the organisms of decomposition and fermentation fill only one part of this world of tiny creatures. Others build up. The nitrifying bacteria, for illustration, oxidize the simple ammonia gases set free by decomposition and the free nitrogen of the air to nitrates—salts which farmers at considerable expense add to the soil as fertilizer. These humble bacteria seem to do this easily, though only recently have our best chemists succeeded in rivaling them and oxidizing the nitrogen of the air in a practical manner. Other micro-organisms have more aristocratic duties than these. The flavor of cheese, of butter, of tobacco, the result of the various "ripening" processes, the fermentation of beers and the rising of bread—all these are for the most part due to micro-organisms.

To go further. We are the hosts of myriad of these little creatures which live especially in our intestine; it is doubtful if we could well get along without them.

In this vast host of micro-organisms, of tiny plants, animals, and bacteria, there are many species, races, and varieties, of which over three thousand are so well known that an expert can recognize them as correctly as you can pick out, in a flower garden, the roses from the lilies, and among the roses can distinguish the tea rose from the moss rose, and, among the tea roses, the Jacks from the Richmond Reds. Of these three thousand known organisms, the most are harmless to man or even useful to him, but there are **between thirty and forty which are decidedly poisonous** to him and which we should try to exterminate. These are the so-called **virulent** parasites, micro-organisms which live at the expense of a host and are **pathogenic** since they injure him. We draw as sharp a line between the pathogenic organisms and the others as is drawn between an edible mushroom and a toadstool. Some of them, as the germs of tuberculosis, are true

parasites—that is, they can live only in and at the expense of a host. Others, as the germ of cholera, are parasites when occasion demands, but they also can live independently—that is, they can support themselves when no host is at hand. These bacilli multiply rapidly under favorable conditions but die promptly when their living conditions (food, heat, moisture, sunlight) are less favorable. Some, however, as the tetanus germ, develop resting forms called spores which can remain almost indefinitely in soil and withstand extremes of heat, dryness, etc., but which, when they find themselves in the tissues of a man, return to their vegetative form and cause disease.

Of these few **pathogenic,** that is, **disease-producing, organisms,** some belong to the plant kingdom. Of these we shall speak on page 512. Of the pathogenic bacteria, we have considerable accurate knowledge, since they are so easily studied. Concerning the pathogenic animal micro-organisms, however, described in detail on page 519, our knowledge, while ever increasing, is scanty, since we cannot as easily grow them in our laboratories, keeping them and their descendants alive for years, as we can the plants and the bacteria, yet we know that they form a very important group, since it is more than probable that some of our worst diseases are due to them.

In addition to these pathogenic micro-organisms which we can see through the microscope, there are others so small that we know that we never shall be able to invent a microscope powerful enough to make them visible, since vision uses light waves and even light waves are too coarse to reveal these tiny creatures. We know them only through their effects. Some of them, however, do pass through stages which are visible, some, like the germ of infantile paralysis, seem to produce structures which can be seen. We speak of these tiny germs as **ultramicroscopic parasites or filterable viruses,** since they will pass through the pores of a clay filter through which no visible germs could pass. It is the study of these

forms, which constitutes the most interesting chapter of the bacteriology of today.

Pathogenic micro-organisms are popularly called **germs.** Some persons still speak of the "germ theory of disease" as if there were still grounds for a difference of opinion as to whether germs really do cause certain disease or not. Not many diseases are caused surely by known germs, but there are a few diseases which we are just as sure are caused by the germs we know as we are sure that the little red, itching spot on our skin was caused by the mosquito which we killed in the act of biting, or that those very painful papules on our skin were caused by the hornets which we saw swarm from the nest we disturbed. The proof that a germ causes a disease is that in every case of this disease the one germ can always be found by the man who knows where and how to look; this germ and many generations of its descendants can be grown in glass tubes in our laboratories; and it, or its descendants, will reproduce that original disease in a man or in certain animals (of course all animals, as man, dog, rabbit, etc., are not susceptible to the same germs) into which it is injected accidentally or purposely (Koch's laws).

The simple presence of these germs, however, does not cause these diseases. It is the poisons which they produce that make the trouble. Each germ produces one, or more, poisons which are peculiar to it, that is, which no other germ can produce. These poisons are called **toxins,** and, since they are peculiar to certain germs, they are called **specific toxins.** Some of these toxins, as that of the diphtheria bacillus, can be obtained separate from the germ, but others, as that of the pneumonia germ, cannot be separated from the body of the organism. It is safe to assume that, if the toxin of each germ could be obtained separate from its organism, the cure of some of the diseases they cause would ultimately be as satisfactory as is now that of diphtheria.

Various pathogenic germs frequently gain access to our bodies, but our body fluids destroy them. If, however, the germ succeeds in disturbing our tissues in any way, that is,

in causing any symptoms, we are said to have an **infection.** If the germ can be demonstrated in the blood the condition is called a septicæmia. If the germ of a septicæmia is pyogenic and causes several abscesses in the body, the condition is called a pyæmia.

Infection. When a germ lodges in any organ, and multiplies there—we have, where it settles, **a local infection.**

In the case of those diseases surely caused by specific germs, the tendency now is not to make a positive diagnosis of this disease until the germ is found or we have other unquestionable evidence of its presence.

That route by which an infecting organism gains access to the body is called its **portal of entry** (see page 394). Some enter through the tonsils, others the bowel wall, etc. Once in the body, some germs have favorite tissues in which they are especially apt to settle, that is, to infect. The typhoid germ selects especially the intestine; the tetanus germ the nerve cells; others the brain, some the heart valves, diphtheria toxins certain motor nerves, etc. Wherever a germ settles it produces its **local lesion,** as distinguished from any diffuse lesions which its toxins alone may produce.

The symptoms of an infection may be limited to **the local lesion,** as the pain of a pimple, or may be both local and also **general,** as the fever, headache, and malaise which accompany a painful carbuncle. Some infections produce no demonstrable local lesion. This is true of the severe forms of blood-poisoning, as streptococcus septicæmia. If while the body is suffering from an infection by one organism another also successfully infects the patient, the second invasion is called a **secondary infection.** This second invader may be a relatively harmless organism which could not have entered had not the first organism robbed the body of much of its resisting power. When this secondary infection causes death, it is called also a **terminal infection.** Terminal infections are responsible for most deaths. This explains why few persons "die of the disease they have," for their serious chronic disease, which is causing a slow, lingering death, is suddenly

robbed of its victim by a more acute terminal infection, which in a few hours, or days, proves fatal.

Certain symptoms **are common to many infections,** as fever, headache, general malaise, etc. But, just as the symptoms produced by various drugs—opium, strychnine, etc., suggest the drug causing them, so there are **symptoms caused by the various toxins** (which also are in fact drugs) **which are individual to the toxin.** The toxin of typhoid fever, for illustration, stupefies the brain; patients with streptococcus infections have unusually clear minds; while those with tetanus are abnormally alert and suffer acutely from even a slight noise.

Any injury to a tissue, whether mechanical, as by a blow, or chemical, as by a poison (and the toxins of the pathogenic bacteria belong in this group), **arouses a response on the part of the tissues** which is defensive, protective, and healing in character. The best known example of such response (or, better named, reaction) is inflammation, with which all are familiar. This is well illustrated by the sore, red, edges of a cut, the pimple or boil at the seat of a local infection, etc. The seat of ordinary infections is indicated by **swelling, redness, heat, and pain,** all due to the increased blood supply to the wounded part, and, later, by the presence of a fluid (an exudate) poured out from the capillaries of the infected area. This, if watery, is called **serum** and if milky (because of leucocytes) is called **pus.** Undoubtedly the purpose of this exudate is to wash away the toxin. Sometimes, as in tuberculosis, in addition to a liquid exudate, the infected tissue **throws up a real wall of new and solid tissue** around the point of infection, producing a **tubercle.** As a rule an infection causes the death of many of the tissue cells located at the point of infection. If these are the important cells of glands, then their place will, as a rule, be taken by worthless fibrous tissue. This explains the permanent scar left by a boil, and also why the muscle walls of the blood vessels, and some glandular organs such as the liver, the kidneys, etc., become, after years of steady, though slight inflammation, transformed into hard

fibrous organs. This sclerosis (hardening) of these organs is not the disease, but the result of healing, i.e., of repair of the areas of killed tissue.

An infectious disease, as explained on page 389, is **one caused by a pathogenic micro-organism.**

Some diseases are due always to **one and the same parasite.** These are called **specific diseases,** and the parasite of each is its **specific organism. Among the specific diseases caused by bacteria** are: acute lobar pneumonia, typhoid fever, epidemic cerebrospinal meningitis, cholera, lockjaw, glanders, tuberculosis, anthrax, influenza, erysipelas, diphtheria, and a few others. **Among the specific diseases caused by animals** (protozoa) are malaria, African "sleeping sickness," amœbic dysentery, lues,* yellow fever,* and a few tropical diseases. **The plant parasites** (yeasts and moulds) cause many superficial inflammations, as thrush of babies' mouths, and trivial infections of the nose, mouth, ears, etc.; some cause serious skin diseases (blastomycosis); some cause lung infections often confused with tuberculosis (pseudo tuberculosis); while others cause true septicæmia. **Some diseases,** as a boil, a carbuncle, arthritis, acute endocarditis, empyema, peritonitis, **can be caused by any one of several germs.** Inflammations with pus production are caused by a group of germs called pyogenic, or pus-producing, organisms (the staphylococci and streptococci especially).

Some of the infectious diseases are definitely **contagious,** or to use the better word, **communicable. A communicable disease** is one which renders the patient dangerous to his neighbors since they may "catch" this disease from him. The better we understand these diseases, however, the less dangerous to his neighbor should the patient be. For illustration, fifty years ago yellow fever certainly seemed an eminently "contagious" disease, so rapidly did it "spread." It seemed due to a "cloud (miasm) which settled down upon a community." Now we know that a patient with yellow fever cannot directly impart his disease to another person, no matter how intimate their relationship. It is spread by

* Recent opinion is that these two organisms should be grouped with the bacteria.

one definite variety of mosquito (page 534). Protect, therefore, a patient with yellow fever from mosquitoes, and his disease cannot spread to others. A similar story is true of malaria (page 526), except that the mosquito in this case is Anopheles maculipennis. Bubonic plague (page 515), which spreads rapidly, killing hundreds of thousands, is not spread by the patients with this disease, but by the fleas of infected rats which live in the houses of these patients. Typhoid fever formerly seemed to be a very contagious disease, but now we know just how to prevent its spreading (page 400). That is, diseases are not as contagious as man had thought, but there is, in the case of each communicable disease, **some one definite danger** to be avoided in order to prevent its spread.

Specific infectious diseases, though they differ much in detail, **have many points in common.** These we will now, in order to avoid repetition, discuss for all.

First, in order that a pathogenic organism may cause disease, **either it or its toxin must first gain access to the inner tissues of the body**, either by penetrating the skin or one of the mucous membranes, or by growing on a mucous membrane, as does Bacillus diphtheriæ, on the surface of the throat, while its toxins are absorbed through this membrane into the circulation. The spot where the organism or its toxin gains admission to the body is called its **portal of entry.** Important portals of entry are skin wounds, as cuts, punctured wounds by nails, as in the case of tetanus, the bite of an insect, etc.; the mucous membranes, as those of the nose, the margins of the gums around dead teeth, diseased tonsils (especially for streptococci), and the bowels wall as for Bacillus typhosus, Bacillus tuberculosis, etc.

And, second, in order that an organism may cause disease, **it must find the potential patient susceptible.** The mere entrance into the body by a pathogenic organism does not necessarily mean infection. Susceptibility to infection will depend, in part, on the virulence of the organism but more on the immunity of the patient to that particular germ. If he

is immune, then the germ cannot gain foothold. If he is not immune, then infection is likely to follow. Susceptibility is both a general and an individual matter. Some persons are fairly susceptible to all diseases, some very little so; some persons are very immune to some diseases and very susceptible to others. We often **are very susceptible to disease we never have had**; we often are **very unsusceptible** to diseases **we have had,** although in the case of a few diseases one attack may make us more susceptible than before to this disease. The person who has had measles, mumps, typhoid fever, smallpox, etc., may expect never to have these same diseases again, no matter how often he may be exposed to them. Through one attack he gains an immunity against that one disease which usually protects him for life. On the other hand, one attack of pneumonia seems to make the next attack easier to get. There is much evidence that we can gain immunity against some diseases, as scarlet fever and diphtheria, without ever knowing that we have ever had those diseases. If immune, the chances are we did have it; but didn't know it. The immunity we gain, however, from a light attack is just as good as if we had had a severe attack.

The importance of susceptibility may be illustrated as follows: formerly we thought that man, in respect to contagious diseases, resembled a soldier. If the soldier on the field of battle gets in the way of a bullet he gets hurt, and the severity of the wound depends much on the character of the bullet. If, later, he gets in the path of a second bullet, he gets hurt a second time, etc. So we thought that a man who in this world "gets a germ" gets an infection. Now we know that we do "get in the way" of germs very, very, often but that nothing happens. That is, in order that something may happen, though the germ must be there, our susceptibility to it is even more important than the presence of the germ.

And, third, the degree of severity of any one infectious disease depends on the susceptibility of the person infected as well as **on the pathogenicity of the germ involved.** Patho-

genicity, in other words, while it is to be measured much in terms of susceptibility is not entirely so, for any one variety of germs may be very toxic or attenuated.

Those infections which run definite courses marked by characteristic symptoms are popularly called **diseases.** For illustration, one might hesitate to call a crop of pimples a disease since the pimples are, so far as the patient knows, the whole trouble; but surely smallpox, which superficially is merely a crop of pimples, is a disease, and the difference is that this crop of pimples is only one part of a much greater trouble. It was an old custom to name diseases after some one striking symptom, e.g., scarlet fever, typhoid (clouded) fever, measles, tetanus, smallpox, ague, etc.

Once the toxins of certain organisms have entered, the body begins the **production of antitoxic substances** which are **just as specific as is the toxin to which it is a response.** That is, the toxin of diphtheria, for illustration, differs from the toxins of all other germs, and the antitoxin which our bodies form to neutralize this one toxin is an antidote for this and for no other toxin. Thanks to these antitoxins, and to the other protective measures by means of which our bodies defend us, we usually recover from infection. This is what we have in mind when we say that an infectious disease **tends to be self-limiting,** that is, that it "runs its course," which means that thanks to the immunizing power of our body, we usually recover spontaneously.

Those germs which gain access to the body are swept by the blood stream over the body, coming into contact with many tissues, but each germ would seem **to have a certain affinity,** or predilection, **for one tissue, or for a few tissues;** to state it differently, certain tissues seem to be particularly susceptible to certain germs, since it is in them that these germs tend to localize. For illustration, typhoid fever always begins as a septicæmia, but in a few days these germs disappear from the blood stream and are found chiefly in the lymphatic tissue along the bowel. Epidemic meningitis begins also as a septicæmia and ends as a local infection of the

membranes of the brain. Tetanus and hydrophobia, on the other hand, are spread along the nerves. In all of these diseases, however, the infection is seldom limited to any one tissue.

Once the germ of a specific disease has gained foothold in the body the latent **period of incubation** begins, during which the germs may be multiplying actively but the patient feels as well as ever. The length of this period of incubation differs much in various diseases, but is, in general, almost the same for the cases of each, although the more susceptible the individual the shorter will be this period. In pneumonia, for illustration, it lasts but one day; in typhoid fever it averages about ten days; in lues it lasts about six weeks. This latent period over, the first definite symptom appears. **The onset** may be very abrupt, as in pneumonia and scarlet fever, or more gradual as in measles and typhoid fever. When gradual, the first symptoms, which may be very indefinite, are called **prodromal symptoms.** In all the prodromal periods the patient feels a "little sick all over" (malaise), but in each of them, there are certain features which are especially suggestive. The child with measles, for instance, may for five days have a little running of the nose, a little smarting of the eyes, and "feels a little mean"; also Koplik's spots, which are very characteristic of measles, appear in the mouth two days before the skin rash appears. The case of typhoid fever has a little headache, perhaps a little stomach ache, and perhaps a nosebleed, etc.

At the end of the prodromal period, **the characteristic illness itself begins.** If measles, the characteristic rash breaks out; if whooping cough, the child, who for a few days "has had a cold in the head," begins to whoop; if typhoid fever, the symptoms characteristic of this disease appear. The laity and the public health officials date the onset of an attack from the appearance of these characteristic symptoms. This, however, is unfortunate. "Scarlet" fever, for illustration, is an old name for a disease which need have no scarlet rash; the disease "measles" is probably spread more easily

during the four days before the child has the rash of measles than after; and there is reason to believe that a patient with influenza spreads his disease to his neighbors during the two days before his own first symptoms begin.

All of the specific infectious diseases are "fevers." That is, **one of their most important symptoms is an elevation of the body temperature,** and the form of their "temperature curve" is one of the best single means of diagnosis and prognosis. So conspicuous is this elevation of temperature that formerly "fever" was considered to be the disease itself, rather than one of a complex of many symptoms of an infection. The words slow fever, continued fever, intermittent fever, remittent fever are still used as the names of diseases. **Fever,** and practically **all of the symptoms which the patient complains of,** are, we should say in passing, evidences of the defense of the patient's body against the invading germs, rather than of the offensive attack of the germ. In other words that which we call the "disease" is the patient's side of a biochemical struggle. This fact needs to be emphasized, in order to enforce the general rule that in our therapy we should not attack the fever itself for fear of weakening the body's own defense. We should try to make the fever itself unnecessary.

The temperature curve of a case of acute lobar pneumonia, of typhoid fever during the second week, of some cases of tuberculosis, and of some of æstivo-autumnal malaria, is elevated and is an almost straight line. Such is called a **continued fever.** Other temperature curves are a succession of sharp rises, and falls to normal. This is called **an intermittent fever.** Tertain and quartan malaria are the best illustrations of this. In them the febrile paroxysms occur at very regular intervals; but of some cases of tuberculosis ("hectic" cases) and of some diseases with pus formation the temperature, while definitely intermittent, is irregularly so. If the fever falls at regular periods but does not quite reach normal, the fever is said to be **remittent.** This is the rule in pus infections.

Most rises of temperature **are attended by an increase, also, in the pulse rate and in the rate of respiration.** In most fevers these rates increase according to certain definite rules; that is, the pulse rate increases ten, and the respiratory rate five per minute for each one degree of temperature. These rates of increase are also a part of the "fever" picture. **Variations from this general rule are important in diagnosis.** For illustration, in typhoid fever and in yellow fever the pulse rate remains almost normal even when the temperature is high, while in acute lobar pneumonia both pulse and respiration usually are faster than would accord with the above rule. The other features of the infectious diseases, e.g., stupor, delirium, skin rashes, enlargement of the spleen, blood changes, etc., are individual to the separate diseases and will be considered in connection with each.

Typhoid Fever

Typhoid fever is an acute fever due to Bacillus typhosus, the characteristic anatomical lesions of which are hyperplasia and later necrosis of the Pyer's patches, and which is characterized clinically by its long course, its continued type of fever, the peculiar rose spots, an enlarged spleen, a slow pulse, and leucopenia.

Nurses should know much about the treatment of typhoid fever since the care of the individual case is in large degree a nursing problem, and since the nurse well trained in the care of this disease is excellently trained in the care of fevers in general.

Typhoid fever formerly held fourth place in our mortality list, killing every year over 35,000 individuals, chiefly young persons under 35 years of age, and each year rendering invalid for weeks and months 400,000 others. In our Spanish-American war, one soldier in every five (there was 20,730 cases) had this disease and of these 1580 died. Yet this suffering and death were needless. The Japanese, who had suffered much from it during the Chinese-Japanese war of a few years before, were able to report but 133 cases during the seven months of the Russian war, while, thanks to prophylac-

tic vaccination, there were between Sept. 1, 1917, and May 2, 1919, in the entire American Army but 213 deaths due to this disease.

Bacillus typhosus (Fig. 128) is really a "distant cousin" of the colon bacillus, an ordinary saprophyte which is always present in our intestines. Yet the typhoid bacillus is not the colon bacillus any more than the deadly toadstool, Amonita, is the same as the similar but highly prized edible mushroom. Between these two organisms are several "cousins" that may cause disease. Some resemble more strongly the typhoid bacillus and are called **paratyphoid bacilli**; others resemble the colon, and are called **paracolon bacilli.** Though Bacillus typhosus is a parasite, it may live for some time outside the body. This fact is important, since only the scientist knows how much sewerage ultimately finds its way into our food and drink. Deficient sewers are a great danger in cities, and in the country cesspools find ready underground connection with wells. Often a single case of typhoid fever living near the head waters of a city supply has infected a whole city. Sometimes **oysters** grown at the mouths of rivers seem to harbor these bacilli, but that danger is not great. **Frozen in ice,** they may live on for several months. The dirty feet of **flies** are much to blame, for they, if opportunity presents, will impartially feed on a manure pile and then in a few minutes light on our food. The **dust** that blows into our houses is also to blame, for Bacillus typhosus can survive in street dust for even 70 days. We "do not wish to know" how **dirty are the hands** of the many individuals who handle our green vegetables, or how filthy are those of the laborers on some little out-of-the-way farm that daily sells a few cans of milk to the agent who collects for some distant city. One such dairy man caused an epidemic of over four hundred cases. Unfortunately, Bacillus typhosus does not change the appearance of milk, cream, or butter, as do the harmless saprophytes which sour it, but which do us no harm.

These germs, however, are not spread by the sick alone but by many who have typhoid fever and get well, for it may

live on for years in their urinary bladder or gall-bladder. Such persons are called **disease carriers.** Their typhoid germs cause them no trouble, but they may communicate this disease to some one else. Typhoid Mary for illustration, a cook in New York, caused 28 cases in the seven families for whom she worked.

Typhoid fever (also the paratyphoid infections) can in large degree be **prevented by the prophylactic vaccination** of well persons with the dead organisms of these diseases. One cubic centimeter of the triple vaccine usually used contains one billion typhoid bacilli and seven hundred and fifty million each of paratyphoid A and B, the most of which bacilli are killed by heat at 53° C for thirty minutes and the others by 0.5 per cent phenol. The first injection for an adult is one half a cubic centimeter, and the second and third, one centimeter each. These injections are given subcutaneously, at one or two day intervals, best at night. Such vaccination has reduced the incidence of this disease in armies to almost one two-hundredths its former prevalence. (In the American Army, the reduction has been from 6.74 to 0.04 per 1000 of mean strength). This means of safety should be enforced in all hospitals, in schools and generally in the community. It is, however, no substitute for good sanitation, for the immunity produced can be broken down by massive infection.

In America typhoid fever is most common in October, and least so in June. Young adults especially are attacked.

Bacillus typhosus enters our bodies by the mouth through the medium of "fingers, flies, or food" and penetrates the walls of the gastrointestinal tract. In the body, it multiplies rapidly, vast numbers are carried around in the blood stream for about ten days, then it disappears from the circulating blood (that is, the septicemia ceases) and its local infections begin. The most common seat of its **local infection,** and possibly a constant one, is the **lymphatic tissue along the intestinal wall.** The larger masses of lymphatic tissue, broad, thin, flat sheets called **Peyer's patches,** are most numerous in

the mucous membrane of the lower two feet of the small intestine, just above the ileo-cæcal valve. The smaller masses, no larger than the head of a pin, are called **solitary follicles.** In a case of typhoid fever the **Peyer's patches** (Fig. 130), instead of remaining about as thick as writing paper, **swell** and in eight or ten days are even an eighth of an inch or more thick. The solitary follicles along the colon wall and the mesenteric lymph nodes also swell, the latter forming palpable masses. Soon the blood vessels of the Peyer's patches become thrombosed and the **mass of tissue dies, sloughs out,** leaving in the mucosa of the bowel wall **an ulcer** which may extend also into the muscularis and even perforate the entire bowel wall causing peritonitis. The solitary follicles suffer in the same way, but are so tiny that they matter little.

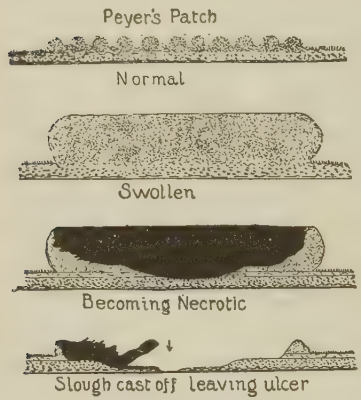


FIG. 130.—The characteristic lesion of typhoid fever. (Magnified slightly.) In this fever the Peyer's patches first swell, then this swollen tissue dies and is cast off as a slough, leaving an ulcer. The arrow indicates a point where the bowel wall may perforate and peritonitis result.

After all the necrotic matter has sloughed away and the ulcer is "clean," healing begins, and soon no trace is left of these deep ulcers.

It is important that the paratyphoid and paracolon bacilli may cause a fever which is similar to typhoid, except that the intestinal lesions are absent or at least are atypical.

The incubation period of typhoid fever is from five to fourteen days, the average, ten. After this period comes a week of **prodromal symptoms.** The patient feels badly, has some headaches, and perhaps, a nose-bleed. He is usually constipated, and the tongue is thickly coated. Soon, usually it is hard to say just when, the **temperature begins to rise,** and the patient feels so weak and miserable that he goes to bed. The temperature continues to rise slowly by a steplad-

der ascent and, in from three to seven days, reaches its highest point, usually from 104° to 106° F. Then it remains constant for a week or more, after which it becomes irregular, a little lower each morning, not quite so high in the afternoons, its curve resembling a "snake fence." This gradual fall continues for a week or more until finally the highest afternoon record is normal. The duration of the fever is very variable. Some patients are well in two weeks, the majority in from four to six weeks, while in other cases (we refer to those without relapse) there is fever for even eight weeks or more.

After the fever has gone **convalescence** begins. The patient is at first thin and weak but he slowly returns to good health or to even better health than he formerly had.

The pulse of a strong man with typhoid fever is usually **remarkably slow** (between 80 and 90) considering the height of the fever (page 399). It is so "soft" that one **feels the dicrotic wave distinctly** enough to mistake it for a second beat. During convalescence the pulse-rate is often **much slower still** (between 40 and 50).

For the first few days of the fever **the headache** is very severe, sometimes terrible. There is **pain in the back** and limbs also. In about ten days from the onset, as a rule, these disappear and the patient, now **dull and stupid,** that is, "typhoidal," does not suffer at all. Then he may be delirious, but this seldom is a serious feature. When very toxic he lies quiet, the mouth open, the tongue dry. It is very hard to rouse him. There is considerable tremor of his arms when he moves them (subsultus tendinum) and he may pick at the bedclothes (carphology) which is a very bad sign. At first he may vomit a little and have diarrhœa or constipation, but vomiting soon ceases. Diarrhœa is always an unfortunate condition, for the patient who remains constipated usually does better. Sometimes the abdomen becomes distended with gas (meteorism).

At the end of the first week the little **rose spots** characteristic of typhoid fever appear on the body, especially over

the abdomen. These are very low papules, rose red in color, and disappear entirely on pressure.

The spleen soon gets large. The **leucocytes progressively decrease in number** and usually are below 5000 (normal 7000 to 10,000), sometimes even 1500 per cubic millimeter.

Typhoid fever is a self-limiting disease, that is, the body cures itself by developing an adequate immunity against the invading organism. (See page 396.) Once well, the patient is truly well, for second attacks of typhoid fever are exceed-

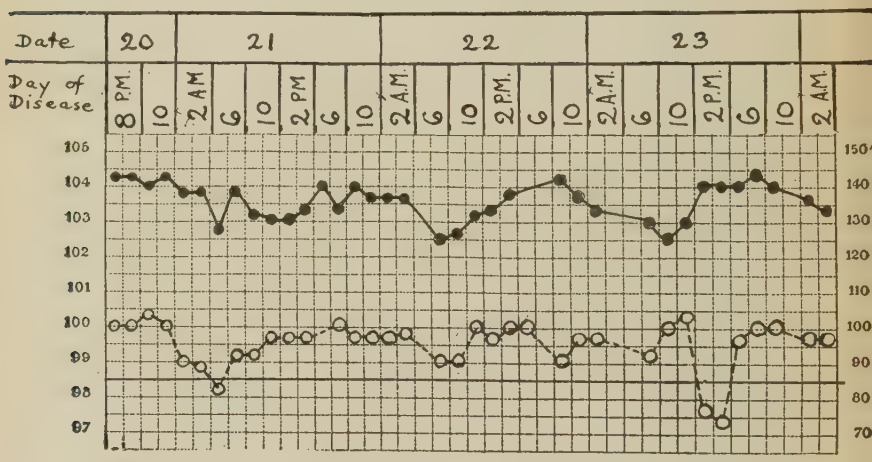


FIG. 131.—Continued fever. Typhoid fever, second week, in a young man.

ingly rare. Often, however, it takes a **long time for this immunity to develop,** and that is why relapses are so common (occurring in ten per cent of all cases). The **evidence of a relapse** is that the temperature, soon after reaching normal, rises again by stepladder ascent and the whole course is gone over a second time, with characteristic temperature curve, enlarged spleen, new rose spots, etc. Relapses are, however, usually shorter than the original attack. A relapse may begin before the initial attack has fully subsided (an intercurrent relapse). Four or five such relapses may follow each other before the patient finally is well, prolonging the disease even to six months. Though such relapses may be reinfections

(from the gall-bladder?) yet the chances are that they are merely evidence that the patient's immunity had not been fully established. In many cases, after the temperature has remained normal for several days, it rises frequently and irregularly. Many of these "post-febrile" rises are due to a **mild cholecystitis**; others, to constipation; some, to an increase in diet; many, to the excitement of seeing friends. Sometimes, and especially in children, the temperature does not for a long time reach normal. Sometimes **several days of**

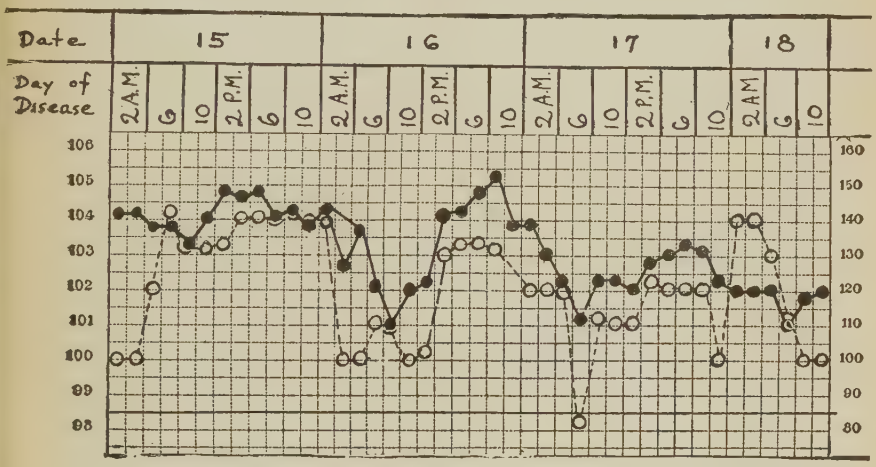


FIG. 132.—Typhoid fever, second week, in a young woman. Note the absence of the typical disassociation between the temperature and the pulse curves. (Compare with Fig. 131.)

hypothermia follow the fever (96° to 97° F.) during which period the pulse is very slow. This period is by some considered a good sign; by others, as evidence of starvation. (See page 415.)

Hæmorrhages from the bowel occur in at least 5 per cent of all cases, and most frequently during the third week. They cause about 10 per cent of all deaths from typhoid fever. Evidently they come from an ulcerating Peyer's patch which, when the slough separates, contains a small artery not yet perfectly thrombosed. Some patients have many small bloody stools, others one or more larger ones, others a sud-

den, fatal hæmorrhage. Of these there usually is no warning, although with the severe hæmorrhages there is a drop in the temperature, the mind becomes clearer, and sometimes the patient has an indefinable feeling of anxiety. Fairly large hæmorrhages illustrate why, a few years ago, bleeding was so popular a treatment for fever. In a few minutes after the hæmorrhage a very ill, delirious patient with high temperature becomes suddenly clear-minded, his temperature falls to normal, the pulse remains slow, and in every way he feels and looks better. The temperature, however, soon returns to its former point, and the fever continues, with but one change, the patient is now weaker because of the loss of blood.

Perforation is the most dreaded complication of typhoid fever; it is the cause of death in almost a third of the fatal cases. When the slough separates the ulcers usually are left with a very thin base, sometimes as thin as tissue paper. In 5 per cent of all cases this perforates, so that the intestinal contents pour out into the abdominal cavity, at once producing a peritonitis which, without operation, is almost always fatal. In the very few cases that do recover without operation, there forms in the abdomen an abscess which later may require operation. Perforation is most common during the third week, although it may occur at any time. Since it is due to almost the same cause as a hæmorrhage, the two often occur together.

Of all the **symptoms** which a patient with typhoid fever may show, none are as important for the nurse to know as are **those of perforation.** Every hour, every minute, after the first symptom, is of great importance, for the earlier the operation the more successful it will be. Hence the nurse should know the danger signals and at once report them to the doctor in charge. The patient's life is in her hands; it is better to give many false alarms than to fail to give the one which may save the patient's life. Our rule is that if a typhoid patient voluntarily complains of a **new abdominal pain, or hiccoughs, or vomits,** this shall be reported at once to the doctor. **The initial abdominal pain is peculiar,** and is one of the most re-

liable of the early symptoms. It is sharp and sudden; the patient sometimes cries out with it; it is different from any pain he has had before; it lasts but a few seconds, then stops, and in a few minutes he may be sound asleep. This last point puts the nurse off her guard, for she can hardly believe that so momentary a pain, which after a few minutes is followed by sound sleep, can be very important. Many patients complain of abdominal pain during the whole course of the fever, but this pain is "different" and is never felt but once. Other early symptoms of perforation also may attract attention; their great value is that they suggest the location of the beginning peritonitis. A rise in temperature followed by a sharp fall is a hint of perforation, yet means little; a slight increase in the frequency of respiration is more important; sudden vomiting, not immediately following nourishment, is suggestive; hiccough, which is rare, but which, when it does occur, even though heard but a few times, is very suspicious indeed of a peritonitis near the diaphragm; and pain on urination suggests pelvic involvement.

Soon after the perforation occurs all these symptoms may cease, and we congratulate ourselves that the alarm was false; and yet even then a peritonitis may be rapidly spreading. If let alone the abdomen of such a case becomes more and more distended; the patient may vomit occasionally for a few hours or days; he looks more and more ill, and gradually becomes weaker until death.

The doctor when first examining a patient with suspected perforation will note **any point on the abdomen which is sore on pressure**; whether or not the leucocytes have risen; whether there is free fluid, or free gas, in the abdomen; whether there is evidence of phlebitis of an iliac vein (for this can cause similar symptoms) etc. The nurse should remember that the earlier the doctor sees the case the easier will be the diagnosis. If he arrives even a few hours late, a diagnosis may be impossible. A general peritonitis in typhoid fever, unlike that of appendicitis, has very few symptoms, probably because the typhoid poison is a narcotic which masks

the symptoms, much as morphia would mask those of appendicitis. With early operation, at least a third of the cases can be saved.

Though Bacillus typhosus enters the body through the intestinal canal, and later produces its most marked lesions there, yet **typhoid fever is a general disease.** During the first week the blood is simply swarming with these bacilli, which may settle in almost any organ, producing symptoms referable to that organ. They may not attack the intestine at all, or may attack it so slightly that this lesion is overlooked. Since the disease affects also other organs early, the first diagnosis is often a wrong one. Some cases resemble at first cerebrospinal meningitis—with convulsions even (these patients say later that they had "brain fever" or meningitis); some begin as an acute pleurisy; others as an acute bronchitis; others with a pneumonia; and still others, an acute nephritis. It is only after these early involvements clear up somewhat, leaving the patient still with a high fever, that the true condition is suspected.

During the course of typhoid fever, **complications are very common.** Some are due to the typhoid bacillus, others to a secondary infection by some other organism which now has a chance since the typhoid infection has so reduced the resisting power of the patient. Bacillus typhosus itself may cause empyema; periostitis (infection of the outer membrane of the bones); venous thrombosis; abscess of the kidneys; ulcers of the larynx; acute endocarditis; myocarditis; pericarditis; pleurisy; acute meningitis; acute nephritis; acute cholecystitis; pneumonia; and infections of eyes, ears, or of the body generally. Examples of the results of **secondary infections** are the terrible bed-sores, the crops of boils, the parotitis, mouth infections, abscess of the liver, etc. **The bed-sores** are sometimes terrible. They occur with the greatest frequency if the patient is not kept clean and dry, or if steady pressure on the back, sacrum, buttocks, heels, etc., is not prevented by pillows and rings and by frequently turning the patient. The skin should be carefully examined every day

and, if any suspicious blue or red spot is noted, this should be kept dry and protected from all future pressure by a ring. And yet in some of the best nursed cases the bed-sores cannot be prevented, since the protective powers of the tissues are so reduced that they cannot resist the common ubiquitous organisms. The mouth should be rinsed out every day and swabbed out repeatedly to prevent mouth infection.

The nurse should watch for any **swelling of one leg** due to **venous thrombosis,** especially of the left femoral vein. If it occurs, an ice-bag is kept over the vein, and the patient is moved with great care and kept quiet for several weeks longer.

The diagnosis of typhoid fever is for the doctor to make, and yet a few points may help the nurse. That a man feels practically well and keeps on with his work does not mean that just then an active typhoid fever may not be in progress, for these "ambulatory" cases are not infrequent, especially among nurses caring for typhoid patients. Nor is the fact that a fever ends suddenly in fourteen days, or less, any strong argument against the diagnosis of typhoid fever, for some cases do this, and without the aid of special therapies. Also, the recurrence of repeated severe chills and sweats is no justification for the diagnosis of "malaria," or "typho-malaria," or "malarial-typhoid," for a true combination of these two diseases is very rare, while the feeling of false security and the resulting laxity in watchfulness, which these diagnoses encourage, are dangerous.

"Typhoid," "typhoidal," "bilious fever," "continued fever," "nervous fever," "low fever," etc., are names of typhoid fever which antedate the discovery of the germ. They served, and still serve, a very good purpose. But the tendency now is to limit the term "typhoid fever" strictly to those cases in which we **can prove the presence of Bacillus typhosus,** no matter what the symptoms present may be. Some cases which for a few days resemble closely typhoid fever are not typhoid, and true typhoid fever presents itself in a great variety of guises. Though a typical temperature and pulse

curve, a history of typical prodromal symptoms, typical rose spots, leukopenia, and a palpable spleen may justify a diagnosis of typhoid fever, yet such perfectly typical text-book cases are about as exceptional as are living composite pictures. Typhoidal is a descriptive term indicating that the patient's mind is clouded by the fever, but some severe septicæmias, malaria, central pneumonia, and some forms of tuberculosis may be definitely typhoidal. The fever is typically "continued" in typhoid fever, but so is it sometimes in cases of tuberculosis, malaria, pneumonia, some abscesses (a small perirectal abscess can closely simulate typhoid fever) and endocarditis. The spleen is enlarged also in malaria. The rose spots when typical are helpful, but some cases of paratyphoid show these. A certain diagnosis is now made in the laboratory rather than at the bedside. The surest test during the first week of the illness is to **find the bacillus itself** in the blood stream. This is done by removing a little blood, with the greatest precautions against contamination (which means careful work by the nurse in cleaning the arm), and by attempting to grow a germ from this. During the first week, in true typhoid fever, Bacillus typhosus can be recovered from the blood of practically every case.

After the first week, this organism is sometimes found in the blood, and also in the urine, in the stools, or in the rose spots. But the easiest method of diagnosis now is the **Widal test.** As before stated, germs produce disease because of certain specific poisons which they produce, and these the body proceeds to control by the manufacture of antitoxins, each of which is an antidote for but one poison. To prove the presence of a definite antitoxin, therefore, is as certain a diagnosis as to find the germ. Of course mistakes are possible. For illustration, a patient now with another disease may recently have been vaccinated against typhoid fever. The antibody we make use of in **the Widal test for typhoid fever** is an agglutinin, that is, one which "clumps" Bacillus typhosus. A few drops of blood of the suspected cases are allowed to clot, and a drop of clear serum obtained. Mix one

drop of this serum with one drop of a fresh culture of Bacillus typhosus and probably every bacillus in this mixture will soon be killed, since our blood is provided with nonspecific substances which protect us against the host of germs which frequently gain entrance to our bodies. But if one drop of this culture be mixed with one drop of this blood serum diluted fifty times with normal salt solution, and within one hour every bacillus stops swimming and all gather in clumps, then we can be sure that that serum contained a special agglutinin (antitoxin) for that particular germ. Sometimes 1/500 of a drop is enough, but the standard technic of the Widal test is, "a dilution of the serum to 1/50 causing in one hour cessation of motility, and agglutination". By this method we can also distinguish between Bacillus typhosus and very nearly related organisms, especially the various paratyphoid and paracolon bacilli.

The convalescence of typhoid fever is long and tedious; it may even take months. The **disease has various sequelæ.** Among these are: cholecystitis, or inflammation of the gall-bladder, which continues the typhoid infection for many (over forty?) years; nervous exhaustion of a very aggravated sort, hence the name "nervous fever"; chronic arthritis of the spine, or "typhoid spine" (painful stiff backs which may last months and greatly worry the patient); chronic neuritis, which makes the patient lame for a long time; and the formation of gall-stones.

The treatment of typhoid fever is largely a problem of nursing. One should remember that without any medicinal treatment, without cold baths, but with good nursing, at least 80 per cent of all cases of typhoid fever will get well; also, that with all that science can suggest in the way of treatment, this mortality of 20 per cent is reduced to 8 per cent. The nurse keeps the patient in such good condition that he can battle to the best advantage with his infection; she is on the watch for any symptoms which may require active interference; and, lastly, she tries to prevent the patient's family and the community from catching the disease.

When planning the **nursing care of a case of typhoid fever** certain features of the disease should be borne in mind; first, that the illness is likely to continue for weeks or even months, and that during the first several weeks of this period the patient will need continuous care both day and night; second, that the mental state of a patient with typhoid fever is one of drowsiness, of indifference to his surroundings, of physical anæsthesia, of partial lack of control of the bladder and rectum; and, third, that typhoid fever is a distinctly dangerous disease to the nurse since the patient's urine and stools are often heavily infected with the germs of this disease, and the patient, because of his typhoidal state, is likely to be very careless and noncoöperative in matters of personal hygiene.

The patient should be treated in a large room from which all but the necessary furniture, and especially all rugs, have been removed. While of course one would choose, if possible, a quiet, well-ventilated and sunny room, yet for a case of typhoid fever these features are much less important than they are for cases of infection of the respiratory tract or of heart disease. Certainly for a case of typhoid fever close proximity to a good lavatory which can serve as service room and large closets for clean linen and other supplies, is of much greater importance. The windows should be well screened to exclude flies.

The bed should be of the right height, narrow, and may well be without a back rest. In these particulars it is the convenience of the nurse, not the comfort of the patient (who will be indifferent), to be considered for she will do harder physical work for a case of typhoid fever, turning him, lifting him, bathing him and changing his linen often, than for almost any other patient. The bed should be well away from the walls in order that the nurse may work easily on all sides, and will need but one thin pillow. Fortunately, active delirium is infrequent, therefore a restraining sheet is seldom necessary, especially if hydrotherapy is well used. The mattress should be new, or newly remade, smooth, and very firm. This is particularly important since a typhoidal patient tends

to lie stupidly and quietly for hours in one position, often with the bed wet or soiled, all of which favors the development of bed-sores, the production of which is much aided by a lumpy mattress.

The **nurse should in large degree isolate herself** with the patient, since she will be continuously in the atmosphere of infection and is not only in danger of contracting the disease

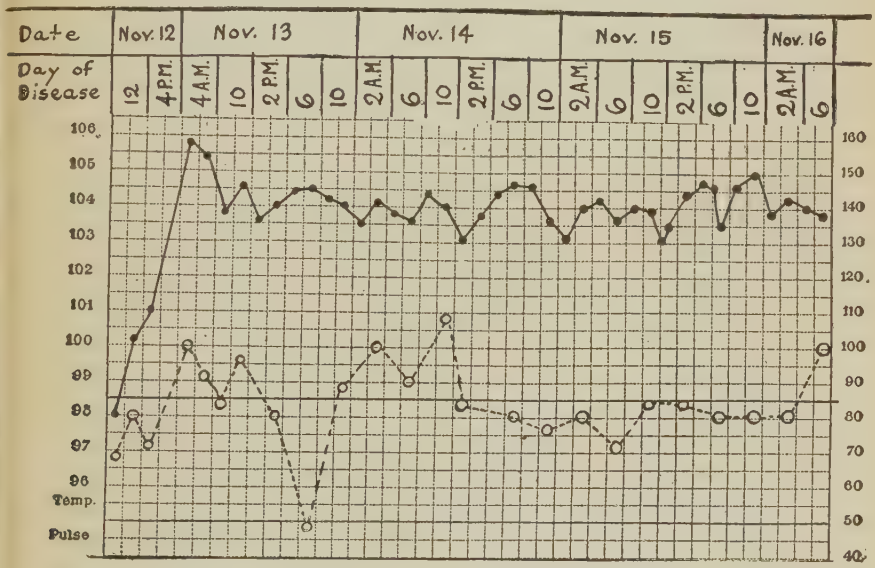


FIG. 133.—Temperature and pulse chart of a case of acute ulcerative endocarditis simulating typhoid fever.

herself but of communicating it to the family. Every dish or glass, all the bed linen and towels, every urinal and bedpan used by the patient are very efficient means of spreading this infection. The urine and every stool should be sterilized (weak carbolic acid is the best) and all the linen used soaked in tubs of lysol before being sent to the laundry.

The routine of the treatment should be outlined early and followed punctiliously. The patient should be aroused for each temperature record, for each feeding, or for his water, and should be methodically turned from side to side in order

to prevent a too continuous pressure on any of the various pressure points of the skin. Since many of these patients are so typhoidal that **they fail to void urine** and as a result the bladder becomes overdistended, reaching even to the umbilicus, the nurse should not be deceived by the frequent involuntary voidings which may mean an overdistended bladder, but should each day measure accurately the output of urine, and if the quantity seems insufficient should ask for instructions concerning the use of a catheter. The rectum also should be watched to avoid an impaction. In cases without diarrhœa a soapsuds enema each second evening should be a routine.

The temperature should be taken by rectum since the patient cannot be trusted to keep his mouth closed while holding the thermometer. At first it should be taken day and night each two hours and a tub or cold sponge given each three hours for a temperature of 102.5° or over. The hygiene of the mouth is quite important, therefore the tongue and teeth should be kept clean of sordes, using swabs moistened with an alkaline mouth wash after each nourishment.

No visitors should be allowed and the relatives should stay but little in the room.

The diet is a therapy of major importance. Four typhoid diets have their friends. Some still believe that milk and egg-albumin diet (the so-called "starvation" diet) is best. Every two hours, when awake, the patient is given either four ounces of milk diluted with two of lime water, or six ounces of lemonade containing the white of one egg. If the milk is not well borne (as shown by curds in the stools) whey, koumiss, or beef broth may be substituted. Ice cream is allowed. Such a diet has a food value of about seven hundred and fifty calories a day. Patients on this regime get their first soft-boiled egg after about seven days of perfectly normal temperature, after which soft food is added daily, and the first solid food is allowed on the 10th or 14th day of normal temperature. It is important that the return to a full diet be very gradual, so that rises in temperature, even relapses, may not result. As such a diet contains only about one quarter

of the necessary heat value, the patient must live off his own tissues, and becomes very emaciated. Since with fever there is anorexia, and always an increased basal metabolism, and since fever is a protective measure, it may be assumed that loss of weight is not entirely an evil.

Diametrically opposed to the above view is the opinion of those who **in addition to liquids give also solid foods**—meat and vegetables—in small quantities. Such solid foods, however, are not as solid when they reach the lower ileum, where the ulcers lie, as are milk and broths. The mortality of the patients thus fed may not be higher (but that is still to be proved), the convalescence may be shorter, but the dangers of sequelæ and relapses are perhaps greater than when less food is allowed. This diet is not really as generous as would at first glance appear, for during the fever the patient eats very little of the solid foods offered him. True, he is very comfortable during convalescence, because he does not feel starved.

Next are those who, in addition to a **soft diet** (two soft-boiled eggs, a little milk toast, etc.) raise, by means of **lactose** or Mellen's food which they add to the milk, the heat value of the food to a point which covers the entire heat loss (at least 2000 to 2500 calories per day for an adult man), and who give solid food as soon as it is asked for.

The fourth plan, and the one which the writer prefers, is similar to the third except that less lactose is used and no attempt is made to prevent a moderate loss of weight.

On one point there is more agreement, namely, that **large quantities of cold water** should be given to the patient, enough to make the output of urine at least four liters a day (some say from 12 to 20 litres. This would mean nearly four glasses of water an hour). In the days of the tub bath treatment, the patient soon found that the more water he drank the fewer baths he got, and so he drank with heroic willingness. The nurses also encouraged him, since each bath required half an hour of hard work. This "internal lavage" efficiently washed out the toxins of the disease.

Hydrotherapy—the cold sponges, cold packs, alcohol rubs, etc., are of great value, **but the cold bath** is a heroic and efficient treatment. The patient is left in the cold water (at from 75° to 80° F.) well supported on canvas strips for twenty minutes, during which time the nurse rubs his arms and legs. After this he is lifted out of the tub, given a cup of hot coffee and is left in the wet sheets covered by blankets for at least ten minutes. These tubs are repeated every three hours if, at that time, the temperature is 102.5° F. or over. One should not, however, follow this rule too closely, for if the patient is very "toxic" a tub is given for a much lower temperature. It is omitted if the patient is suspected of having perforation, or if he has had a recent hæmorrhage, a phlebitis, or great prostration. Often the first bath is given at 85° F., but only to accustom the patient to this therapy. Baths are given, not primarily to reduce the temperature (although after the first week they are followed by a fall), which could be more easily done by drugs, but because fewer patients thus treated die. Among the advantages of the tubs is the fact that they are an excellent tonic to the nervous system. Following them the delirium disappears, the patient feels better, his mind is keener. Because of them the respirations are deeper, the kidneys act more freely, the heart is stronger, and the condition of the skin is better. Unless the clinic must care for several patients of typhoid fever each season it may, however, be difficult to organize tub treatment, and ice sponges are used instead.

The drugs intended to reduce the temperature, to disinfect the intestine, etc., all are of doubtful value. To relieve the tympanites, turpentine stupes are given, or, better, a rectal tube introduced, and left there for some time. **When hæmorrhage has occurred,** or is suspected, the foot of the bed is elevated, the patient moved as little as possible, and nothing given by mouth except calcium lactate, a lead and opium pill, and sips of water.

Since the **urine sometimes contains many typhoid bacilli,**

it is customary to give all patients urotropin, five grains three times a day, each second day for weeks.

The nurse's problems during the convalescence are especially difficult. A patient previously on a low diet often now will steal food. Constipation should not be relieved by drugs, but by enemata, until the patient is quite well again. A back rest is allowed as a rule on the seventh day of perfectly normal temperature, and a chair about four days later. The patients must be guarded against excitement and over-exertion. Relapses are too common and the family, in their efforts to explain them, are apt to blame the nurse.

Pneumonia

Of the acute fevers **acute lobar pneumonia** deserves the most careful study, since it is one of the commonest and most fatal of them all. Lobar pneumonia is caused by **Micrococcus pneumoniæ** (Fig. 127, *E*), or rather by a group of germs which bear that name and of which **there are four types.** Those of Type IV are almost constant inhabitants of our mouths during health, and seem always waiting for an opportunity to invade when the soil of our bodies shall be favorable. These explain 26 per cent of all cases of pneumonia, mild attacks as a rule. Type III is the very fatal Streptococcus mucosus which causes 13 per cent of all cases. Type I causes 36.5 per cent and Type II 24.6 per cent of all cases, and these are the more familiar forms of acute lobar pneumonia.

At the beginning of practically every attack of lobar pneumonia, Micrococcus pneumoniæ is carried by the blood over the whole body, has its choice of practically every organ, and yet usually chooses the lung. To find it in the blood, after the onset, means usually a fatal outcome.

It will be remembered that our lungs are made up of many little lungs (Fig. 54), each a group of air sacs (alveoli) communicating with the outside air through the bronchial tree. In the walls of the alveoli is a network of pulmonary capillaries full of blood. **Pneumonia is an inflammation of the epithelial walls** of these alveoli. The first sign of this inflammation is that the pulmonary capillaries **become very con-**

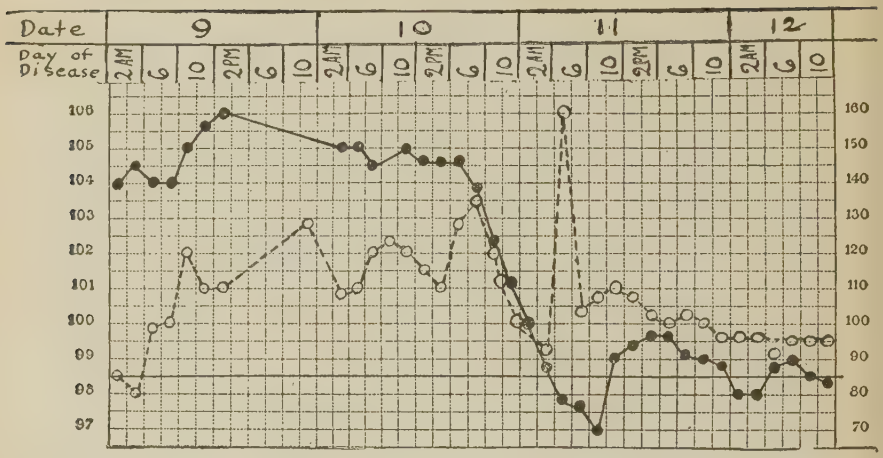


FIG. 134.—Temperature and pulse chart of a case (a male 30 years old) of acute lobar pneumonia terminating in crisis.

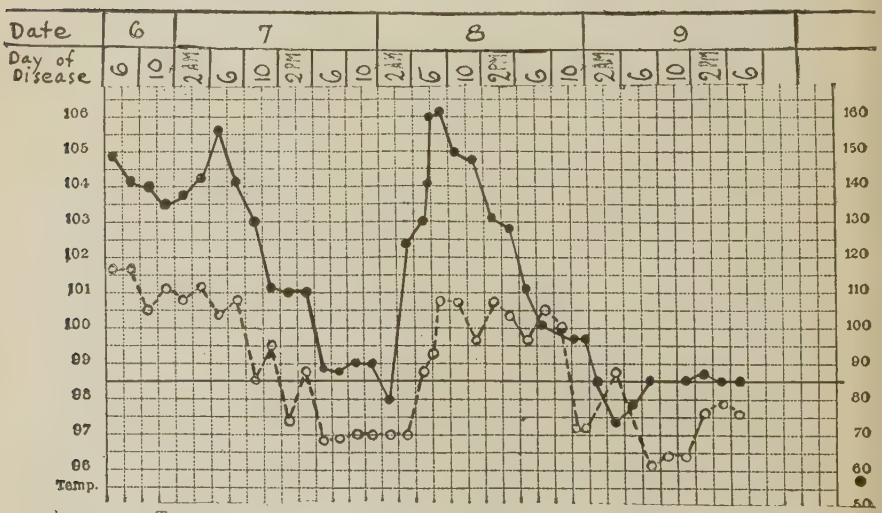


FIG. 135.—Temperature and pulse chart of a typical case (a boy 15 years old) of acute lobar pneumonia with both pseudocrisis and true crisis.

gested and that blood-plasma oozes through the previously water-tight wall of the alveoli into the air spaces. This is the **stage of engorgement.** Then, an inflammatory exudate consisting of serum, red corpuscles, and fibrin is poured into the air-cells, and fills them with a solid red clot (Fig. 136). This is the **stage of red hepatization,** red because of the predominance in the clot of red blood cells; hepatization, because the involved lump of tissue is now no longer a sponge of air-cells, but a solid organ, which feels and looks like liver tissue. Through the capillaries the blood still circulates, but now there is no air in the alveoli to purify it. Next, the red cells in the exudate disintegrate and leucocytes migrate into the clot in large numbers, giving it a gray color, whence the expression, **stage of gray hepatization.** Finally, the leucocytes themselves disintegrate, and by doing so liberate from their bodies a trypsin-like ferment which soon changes the solid clot in the alveoli to a liquid, just as trypsin in the bowel can liquefy a solid piece of meat. This process is called **resolution.** The liquefied exudate is now reabsorbed into the blood (very little is expectorated) and then excreted in the urine. After the absorption is accomplished the alveolus is again full of air and soon is in about as good condition as before the attack.

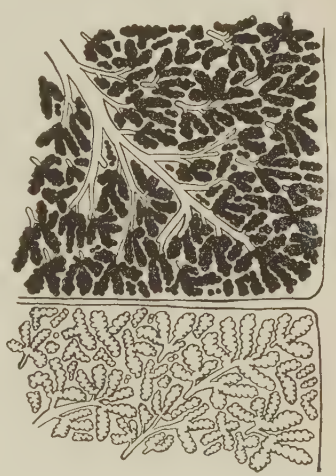


Fig. 136.—The adjacent margins of two lobes of the lung. The lower lobe is normal, the upper is the seat of a lobar pneumonia. Note that the air cells are filled by the exudate, the bronchi are free.

The process which we have described as taking place in one alveolus goes on in all the alveoli of the inflamed lobe, or lobes. The involvement is **by lobes,** whence the name **lobar pneumonia,** a name which distinguishes this disease from lobular pneumonia, a condition in which many isolated groups of alveoli are involved. It is plain to see that since

the alveolar epithelium is a surface membrane, and since the exudate is in the air cells, pneumonia is in fact a surface disease in the same sense that diphtheria is a surface dis-

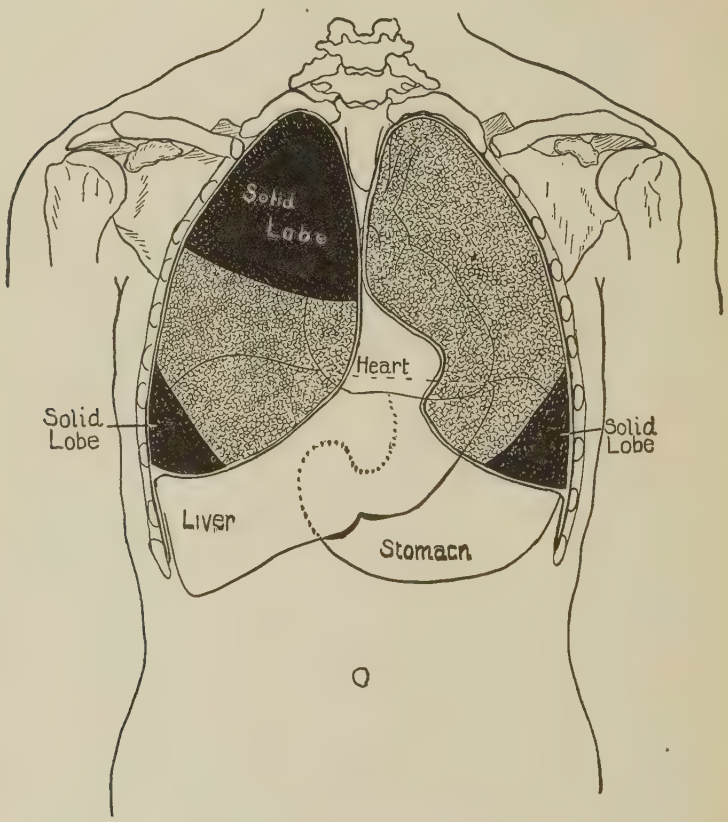


FIG. 137.—Sketch of the lungs of a case of double lobar pneumonia. There is consolidation of the right upper and lower and of the left lower lobes. (Only the tips of the lower lobes can be seen in a front view.)

ease. The real danger in pneumonia, as in diphtheria, is not from the exudate nor from the germs directly, but from the toxins of these germs which are absorbed into the blood stream from this exudate.

The onset of an attack of lobar pneumonia is **characteris-**

tically sudden. In fact no acute disease as often begins with the suddenness of this. Often the patient can tell to within five minutes just when his attack began. A man who has been feeling perfectly well is suddenly attacked by a **sharp pain in the side** which prevents his taking a long breath, then begins a **severe chill,** followed by fever. In no acute disease is the chill at onset so constant or so severe as in this. The patient from the very first feels ill and goes to bed at once. **His temperature rises rapidly,** on the first day reaching from 104° to 106° F. **The cough** begins at once, a frequent short, painful cough; he expectorates a sputum which at first is sometimes clear and sometimes very bloody, but which soon becomes **typically rusty in color and so sticky** that he can hardly expectorate it, and it must be wiped off his lips. So sticky is it that the sputum cup half full may safely be held upside down. He lies in bed often surprisingly flat considering his dyspnœa, but usually is propped up, **breathing rapidly,** his cheeks flushed, his eyes bright, the nostrils **dilating with each inspiration,** and **grunting** with each expiration. The pulse is rapid and bounding. The leucocyte count **ranges after the first day from 20,000 to 100,000 per cubic millimeter.** This is the best illustration we have of an inflammatory leucocytosis and this is a great aid in the diagnosis. **Fever blisters** are common on the lips and nose.

The patient continues in this condition for from three to ten days, the temperature curve the straightest seen; then comes the crisis, and convalescence begins. **The crisis** in pneumonia is a remarkable phenomenon. Up to this very point the patient has seemed to be getting worse, seems to be even at death's door; then, within a few hours, the temperature falls to normal, the pulse and respiration rates become normal, the cough ceases, and the patient is comfortable and practically out of danger. The lungs, however, show very little change. Presumably they at once begin to resolve, but the sudden improvement is not due, so far as we can see, to any change in the lungs. The fever, the rapid pulse, the cough, the sputum, and all the other symptoms, were due to

the toxin which the germs had been furnishing the blood and which the blood stream had carried to all parts of the body. The crisis and the resulting prompt recovery doubtless mean that the protective forces of the body have at last got the upper hand. These protective bodies in pneumonia, however, whatever they may be, are not antitoxin, but all are antibacterial. No antitoxic bodies have as yet been proved. If the patient dies it is because of a toxæmia, that is, because of the patient's susceptibility to the toxin of this germ, and not because of any local physical conditions in the lungs. The constitutional symptoms bear no relation to the amount of lung involved, for a person with but part of one lung consolidated may be much more ill than others, four of whose five lobes are solid from pneumonia.

The mental symptoms in pneumonia deserve especial mention. The patient very often has a restless, excited delirium which requires restraint since he may leap through an open window. There are cases in which the first sign of the attack of pneumonia is a maniacal insanity, the pneumonia at first unsuspected. Alcoholics with pneumonia are often thought to have delirium tremens.

In some cases the temperature does not fall by crisis—that is, within twelve hours, but more slowly, reaching normal in from twelve to twenty-four hours. This is called a **protracted crisis.** If the fall takes over twenty-four hours—even three or more days, the defervescence is called a **lysis. A pseudocrisis,** that is, a rapid fall of temperature, but without much change in the other features of the case, may precede the true crisis. **A true crisis** may come as early as the third day, some say even at the end of the first day (larval pneumonia), but most often it comes between the seventh and tenth days, and sometimes later. As a rule the consolidation resolves rapidly after the crisis, but in cases of **delayed resolution** it may take even six weeks.

If at least one lobe of each lung is solid, the case is one of **double pneumonia.** This form is not more serious than

when two lobes of one lung are involved. If as one lobe resolves another becomes solid, the case is one of **creeping pneumonia.** When in the course of a chronic disease—Bright's disease, for example—a rapidly fatal pneumonia develops, the latter is called a **terminal pneumonia. Traumatic pneumonia** often follows injuries to the chest. It is said to follow a general surgical anæsthesia (ether pneumonia), although the majority of these are **metastatic pneumonias** (that is, pneumonias due to infection brought by the blood to the

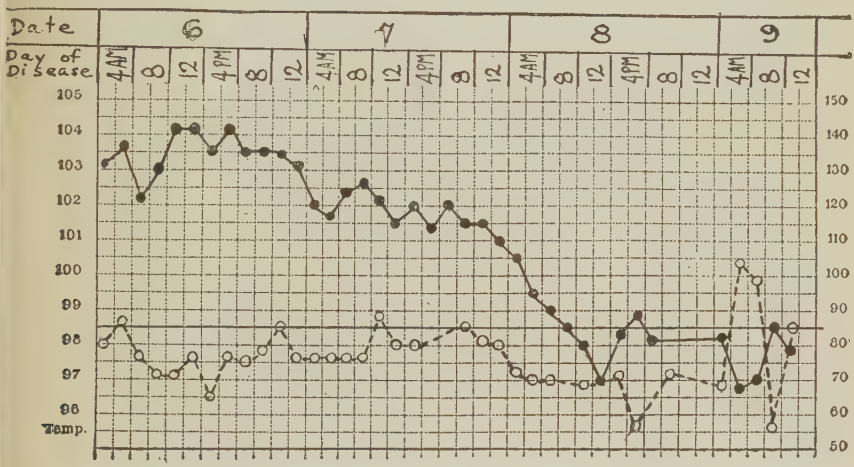


FIG. 138.—Temperature and pulse chart of a case (a male 20 years old) of acute lobar pneumonia terminating in protracted crisis.

lung from the seat of the operation). Although the pneumonic process involves usually a whole lobe uniformly, yet a portion may be spared. This portion is sometimes the surface of the lung, in which case we have a **central pneumonia.** Many cases are central for a day or so, or until the disease has reached the pleura. There is practically always a pleurisy with a pneumonia and this causes practically all the pain since the pleura is a very thin membrane covering the inflamed lung.

As a rule with the crisis or lysis **the danger of pneumonia is over,** for there are but few sequelæ. In some cases, how-

ever, pneumonia is followed by empyema or by abscess of the lungs.

Pneumonia is one of the most serious of all infectious diseases, and yet, leave the cases alone, do practically nothing, even let them want many of the necessities of life, and over 70 per cent will get well. Do as much as we can for them, using all the science at our command, and nearly 25 per cent will die. These figures apply to large hospital clinics, to which all varieties and grades of pneumonia are admitted. Among the well-to-do the mortality is nearer 10 per cent. Nevertheless, a few whose lives are in the balance we do save by tiding them over until the protective powers of the body are sufficient to control the situation—that is, until the patients shall have cured themselves. Of pneumonia it has been said that the young all get well, and the old all die, but there are many exceptions to this rule. Pneumonia is often spoken of as the "old man's friend," because terminal pneumonia brings him a very quick and painless release and saves him from the lingering course of a chronic disease.

Pneumonia is most prevalent in the early spring. It attacks especially the inhabitants of cities, persons of sedentary habits, those suddenly exposed to cold weather (especially half invalids) and above all others, alcoholics. It is very apt to attack a person more than once, even several times. It is a contagious disease and may spread through a whole family, but this is rare.

The treatment is to help the natural protective agencies of the body in conquering the disease. Serums and derivatives of serums have been prepared, all of which have value in the treatment of some cases. Serums are on the market for cases due to the organisms of Type I and Type II, but they still are of limited value. Pneumonia not treated by a serum is a "self-limiting" disease and the outlook in some measure will depend on the skill of the nurses.

The nursing care of an acute case of lobar pneumonia should be carefully organized. The disease is likely to be short, not over a week in duration, but during that time

constant attention day and night is necessary and the startling emergencies which arise are much more frequent than in typhoid fever. Fortunately, the danger of contagion to the nurse and family is not great, yet the sputum, and therefore the sputum cup, handkerchiefs, etc., are dangerous sources of infection and should receive the same care as in tuberculosis. Fortunately the patient, usually mentally quite alert, will, as a rule, coöperate well with the nurse. The happiness and comfort, therefore, of the patient are very important considerations. The room should be large, sunny, and airy. The problems of plumbing, so important in typhoid fever, may well yield to those of sun and air. The fad for outdoor nursing of pneumonia is not nearly so popular as a few years ago, excepting for those patients accustomed to the outdoor life. Since the **delirium of pneumonia often is of the maniacal and violent type,** and sometimes develops rather unexpectedly, the patient should not have the opportunity to leap through an open window when left alone for a few minutes, and a restraining sheet should always be at hand. Even of those patients not delirious the mental state is one of excitement, therefore the patient should receive no visitors, the calls of the family should be brief, and no business details and no news which might excite him should reach him. The bed should have a comfortable back rest in order that during any paroxysms of coughing he may not have to exert himself to sit up. The patient should wear a flannel nightgown and be carefully protected from chilling. The chief effort of the nurse should be to **keep the patient physically and mentally quiet.**

There is perhaps no disease of adults in which **sudden death** is so likely to follow a sudden change of posture as in pneumonia. This is not due to "failure of the heart," but to vasomotor failure. The patient therefore should be kept perfectly quiet, in this way often preventing death by protecting his circulation. Therefore, the nurse should have ever ready a hypodermic syringe filled with cardiazol or adrenalin. He should be assisted even when he turns over in

bed. He certainly should never sit up suddenly, since too often patients who do this fall back dead. If oxygen is to be used the patient's head should be well enclosed in a suitable tent. The funnel on the oxygen tube suspended a few inches above the mouth is of very little use. The air of the sick room should be fresh, or, if possible, the bed wheeled outdoors during the day. **The diet** may be anything suitable for an invalid. **The amount of water allowed** will depend on the condition of the heart. Certainly we should not force fluids as we do in typhoid fever, for when we try we fail. Tepid, cold, or ice sponges are very beneficial and may be given even every three hours. They are an excellent tonic to the nervous system, make the patient more comfortable, and clearer mentally. **An ice-bag or warm poultice** is kept on the chest. Theoretically a hot flaxseed poultice is the best, and practically is often the most comfortable measure. Some prefer an ice-bag, but it should be one large enough to surround half the chest. Its chief object is to relieve the pleural pain. When this is very severe, either a hot-water bottle, the Paquelin cautery, or morphia is indicated. When the pulse is rapid and feeble an ice-bag should certainly be kept over the heart. Salts are daily given to keep the bowels open. Many give digitalis to support the heart.

Micrococcus pneumoniæ may cause other conditions also, such as septicæmia, bronchopneumonia, pleurisy, pericarditis, endocarditis, meningitis, peritonitis, arthritis, otitis, ulcers of the cornea of the eye, and finally a septicæmia without local lesion.

The pleurisy caused by this organism is usually purulent, —that is, an empyema,—which usually requires for its cure the resection of one of the ribs in order that the pus may drain out. Children, however, often get well after the pus is merely removed through a needle.

The endocarditis it causes is usually of a very malignant type. **Pneumococcus meningitis** is a disease especially of children and differs from the epidemic form (see page 458) in that the inflammation extends equally over the whole surface of the brain, and also in that it is uniformly fatal.

Bronchopneumonia

Lobular or bronchopneumonia differs from lobar pneumonia in that in the former disease groups of a few alveoli only and not whole lobes are involved. The areas involved may be the alveoli of a bronchus which is the seat of an acute bronchitis or those surrounding an infected thrombus. A lung thus affected feels, when collapsed, like a soft cushion full of hard lumps, which vary in size from a pinhead to a hen's egg. If these lumps are numerous enough the lobe may be almost completely solid, but its consolidation is not uniform, as in acute lobar pneumonia, since the pneumonic areas are of different stages of consolidation and between some of the areas will be narrow strips of normal lung. This is the form which as a complication makes measles and whooping-cough diseases dangerous for young children. This, also, is the form of pneumonia which made the influenza epidemic of 1917 to 1919 so terrible. It is a serious disease for babies. It is the commonest form of terminal pneumonia in cases of blood-poisoning, meningitis, typhoid fever, endocarditis, etc., and often is the immediate cause of death in these conditions. In chronic tuberculosis the form of lesion is a bronchopneumonia.

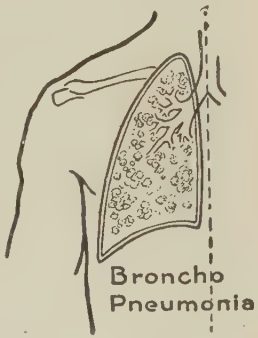


Fig. 139.—Bronchopneumonia.

Unlike true lobar pneumonia, lobular pneumonia may be **caused by a variety of organisms**: some cases, and especially in young children, are pure infections by Micrococcus pneumoniæ, while if two organisms are present, as in typhoid fever, the pneumococcus is one of them. The treatment is that of acute lobar pneumonia.

Tuberculosis

Acute tuberculous pneumonia at first resembles closely acute lobar pneumonia, but of acute lobar pneumonia the cause is Micrococcus pneumoniæ and of tuberculous pneu-

monia it is Bacillus tuberculosis. The toxin of this latter germ is far more destructive than is that of Micrococcus pneumoniæ, for although early the tuberculous pneumonic area resembles one of lobar pneumonia yet later the tuberculous toxin kills the walls of the alveoli as well as the cells of the exudate, so that the solidified area becomes a mass of dead matter. Cut through this, and the cross-section, because of its combination of white, gray, and green colors, resembles some beautiful green marbles. If the patient continues to live this dead mass soon begins to liquefy and to be removed through the bronchi as sputum, **leaving a hole or cavity** in the lungs, the size of which will depend on the amount of lung which has died and on the duration of the disease; for some patients die before softening has begun, while others live till practically none of the diseased lung is left—only a hollow, empty, pleural sac. Some patients recover from the acute illness but the cavity will remain, or, will be slowly obliterated because of the contraction of its walls, until **only a scar is left.**

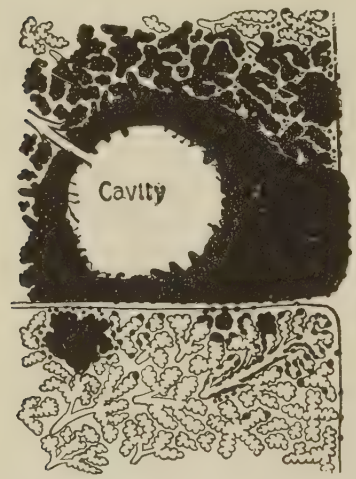


FIG. 140.—This figure represents the adjacent edges of two lobes of a lung. There are miliary tubercles in the lower lobe, and conglomerate tubercles in the upper. In the upper there is exudate, and destruction of air cells and bronchi alike.

Early in the case, tuberculous pneumonia is often confused with acute lobar pneumonia. True, it is a lobar pneumonia but it is not due to Micrococcus pneumoniæ. Early this mistake is almost unavoidable unless the patient is known to have had previously a chronic lung infection, (for tuberculous pneumonia is often an acute flare-up of a chronic tuberculosis). With this diagnosis and too often a hopeful prognosis, all goes well with the patient till the eighth, tenth or even

the fourteenth day of the fever when the failure of a crisis or lysis to terminate it arouses fears that the case was not one of "acute lobar" pneumonia. Soon the sputum becomes

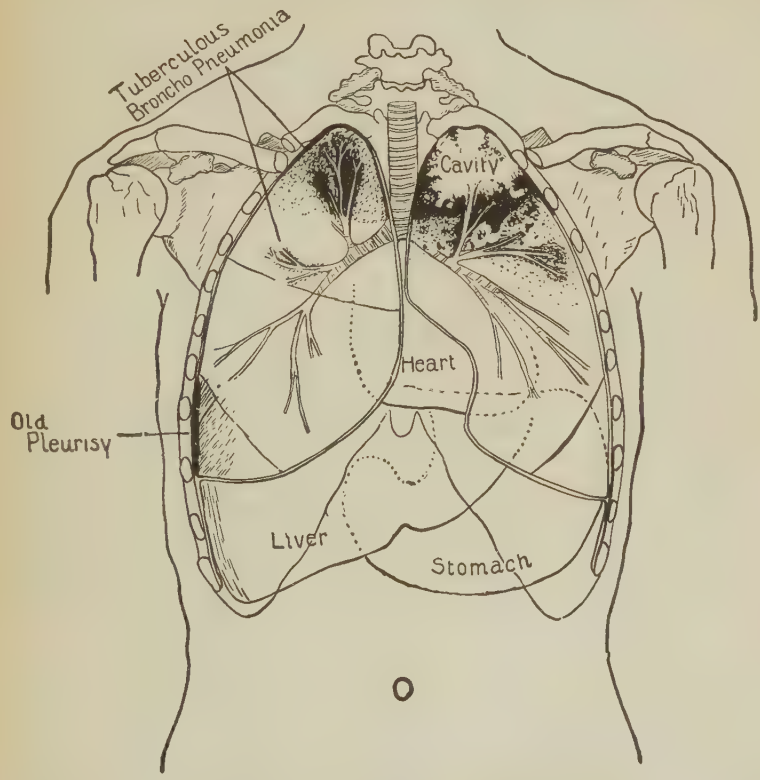


FIG. 141.—A diagram of the chest, showing tuberculosis of both lungs. The oldest tuberculosis trouble is the pleurisy of the right lower axilla. This has healed, but has left a patch of "thickened pleura" and slight fibrous changes in the lung. The most recent lesion is the slight bronchopneumonia at the base of the right and left upper lobes. There is a much more extensive tuberculous pneumonia at the apex of the right upper lobe. At the apex of the left upper lobe is very extensive tuberculous pneumonia, with cavity formation. Scattered areas of beginning bronchopneumonia and scattered miliary tubercles are represented in the lower part of this lobe.

yellower, the temperature more irregular, the patient begins to fail, and sputum examination reveals Bacillus tuberculosis.

A tubercle (little tumor, little hard lump) is the characteristic lesion of tuberculosis, and that from which the dis-

ease gets its name. When a few tubercle bacilli lodge in the wall of an air-cell or bronchus and are not killed by the tissue fluids, the tissue cells in their neighborhood quickly build a wall of cells around them. The result is a little tumor, just big enough to be seen with the naked eye, and called a **miliary tubercle.** Then begins a struggle for existence between the bacilli and these tissue cells. If the cells win and the germs die, then the tubercle remains as a little tumor which later calcifies. If on the other hand the toxin of the germs win then the whole tubercle caseates—that is, dies, and becomes a little lump of cheesy, that is, yellow clay-like matter, the bacilli multiply, new tubercles form in the wall of the old,

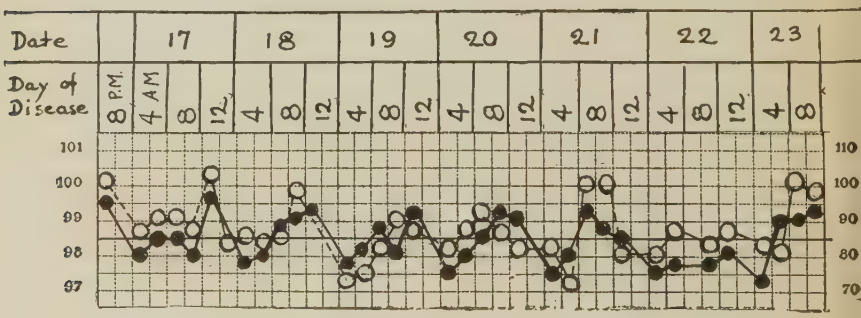


FIG. 142.—The afternoon fever of tuberculosis.

while around them all the lung builds new walls of protecting tissue. If this stops the process, the cheesy matter will soon become calcified—that is, impregnated with lime salts; but if the germs continue to multiply the process continues until large masses of tubercles, even as large as one's fist, called **conglomerate tubercles** are formed, and surrounded by a wall of scar tissue (an attempt at healing). The tissue in the centre of these large masses later may die and the dead matter be expectorated through the bronchi, leaving a cavity in the lung. Since scar tissue always contracts the cavity later becomes much smaller and may disappear leaving a scar, but, quite as often new tubercles start up in this protective wall, and so the disease spreads on. Years later we find in

these puckered scars calcified masses and cavities almost closed by the contraction of their thick walls. If no scars are present the **lymph-nodes along the bronchi** will tell the story, for they will be large, caseous, or calcified masses. One remembers that the lymph-nodes along the lymph-vessels are filters for the tissue lymph, and it is in this tissue that the bacilli live; hence these nodes must bear the brunt of an infection.

Chronic pulmonary tuberculosis, characterized by a slow formation of conglomerate tubercles, and followed either by fibrosis or by slow cavity formation, is the common form of consumption. It is very insidious in its onset and course. The patient usually has been treated for a dozen diseases before the correct diagnosis is made, which is very unfortunate, as it is much easier to cure the disease early than it is later on.

Suppose that a few of the tubercle bacilli lodge in the walls of the smaller bronchi at the apex of one lung,—their favorite location. At once a few tubercles will form around them, a few alveoli be filled with exudate, and a tuberculous bronchopneumonia will slowly spread from this as a centre. What will the symptoms be? In the first place, the patient may not notice a single symptom until the disease has reached a fairly advanced stage. Cases of "early" or "incipient" tuberculosis with "latent onset" are not at all rare; such a case is not "early," nor "incipient," certainly not if the patient is an adult; the term **minimal** therefore is used. The chances are that this patient will notice that he is losing weight, that he fatigues a little easier than before, that he is a little pale, that he has a little indigestion and a poor appetite. He certainly will not realize, until someone tells him of it, that he coughs or at least "clears his throat" every morning just after rising. He may or may not expectorate what is raised, but if he does, it is "only slime," "only mucus from the throat." He will perhaps feel rather tired afternoons between four and six o'clock, but in the morning he feels in fine condition. If by an accident his temperature is taken,

the chances are that it is found to be subnormal (95.5° to 98.4° F.) in the morning and from 99° to 99.5° in the afternoon. "No fever, merely a high normal temperature," he will say. He surely will take some medicine now. If his paleness is marked, a diagnosis of "anæmia" often is made. Then iron in good doses, and some "tonic" are given to him. Perhaps his loss of appetite and his "indigestion" are what trouble him most, and so his stomach is treated. His indigestion may be extreme; he may vomit every meal; he may have severe pain after eating; he may loathe the sight of food. It is very difficult to persuade this man that his stomach is perfectly normal; and yet such is often the case. Or perhaps his trouble began with a cold, which "has hung on." He knows that he does cough a little every morning but the slight amount of innocent-looking sputum does not worry him. To him it is only a case of "neglected cold," of "chronic bronchitis," a "winter cough"—nothing more. Or, perhaps the trouble is a little more advanced than we have been supposing. He has chilly feelings in the afternoon, or a slight sweat at night. The chances are that he will be sure it is "malaria" and will begin taking quinine. Or perhaps he has a slight touch of pleurisy, a "stitch in the side," or a little "water on the chest." He consoles himself with the reflection that it is only "pleurisy." Perhaps he has noticed that he is getting hoarse, possibly it also hurts him to swallow; "chronic laryngitis" is his consoling thought. Perhaps the glands in his neck get large and stay so; yet this does not worry him; "scrofula," he says. Or possibly while "in the best of health" he has a sudden desire to cough, and expectorates a mouthful of bright red blood. Then he is frightened. This man is fortunate, for, although his case need not be as advanced as many cases of the kind described above, he now is ready to obey orders, and so he will have a better chance to get well than any of the others.

There are today in every large city probably hundreds of patients who are treating themselves, or are being treated, for one of the above-mentioned diseases, and yet their real

trouble is a little spot in the lungs, which gives no local symptoms. There the disease is either spreading slowly, or is being slowly controlled by the self-protecting agencies of the body. In over one half of these cases the correct diagnosis will never be made, but the patients will get well, thanks to their own inherited **vis medicatrix naturæ,** and no thanks to any of the expensive tonics they have been taking.

Other patients, however, after a few months during which the diagnosis and hence the medicines have been changed several times, finally realize that they have a lung trouble. The cough has become frequent and troublesome; there is no doubt as to the afternoon fever; the patient has night-sweats; he loses weight and strength rapidly, and he is obliged to admit that he is "going into a decline" or that he "has a chronic cough, which may turn into consumption if he doesn't check it at once." So more cough medicine is taken. Finally, he admits that now at least he has consumption. "Has consumption?" He has had it a long time, even before that day when months ago he first noticed a little paleness, dyspepsia, or loss of weight; but such is the optimism of tuberculosis patients, such the effect of the euphemisms used by their friends, that the disease has had months in which to get good headway—now it claims its victim.

Hæmorrhages are frequent accidents of chronic tuberculosis. Some occur very early in the disease, and they may even be the very first symptoms. Small in amount, these early hæmorrhages are never serious, and they usually serve the purpose of frightening the patient into taking good care of himself. Some cases, the "hæmorrhagic type," have hæmorrhage after hæmorrhage during the whole course of their disease. The most important hæmorrhages, however, occur when the patient has almost or entirely recovered from consumption. These are usually profuse, often fatal, and they come very unexpectedly. They are not due to the activity of the tuberculosis, but to the injury inflicted years before on the arteries during cavity formation. (Page 96.)

When a person suddenly begins **to bleed from the mouth,**

the question of the source of the blood is important. It may come **from the lungs,** as in consumption, or **from the stomach,** as in a case of gastric ulcer. In the latter case it is vomited rather than coughed up, although with vomiting there is often considerable coughing, and *vice versa.* The blood from the stomach is usually dark in color and often mixed with food, while in lung cases it is bright red and frothy. In mitral heart disease, in cases with pulmonary infarction, in bronchiectasis, and in those with ulcers of the larynx, the hæmorrhages may be profuse. In aneurism of the aorta a rapidly fatal hæmorrhage occurs if the aneurism ruptures into a bronchus. There are several other causes of pulmonary hæmorrhage, but these are the most common.

The course of a case of chronic pulmonary tuberculosis is variable. The tuberculous bronchopneumonia may progress rapidly, cavities rapidly form, the fever run high and irregularly and with daily chills, until in a few weeks the patient dies of "galloping consumption." Or, the patient with a slower process and less marked symptoms may feel now better, now worse; now he may be apparently well, now ill in bed. So it goes for months and years, until an acute exacerbation of the trouble, often a tuberculous pneumonia, ends the scene. Sometimes the patient **"gets used to his disease."** He feels well. If a laborer he is able to do a hard day's work and keep it up for years. Yet he has a chronic cough with slight expectoration and is always spreading the disease among those more susceptible than himself. He may live fifteen or more years, and he will probably have an opportunity to attend the funerals of some of the doctors who have told him of his consumption, and have warned him to begin treatment at once. But he will attend the funerals of relatives also, especially of members of his own family, and he will little think that he is directly responsible for these deaths; that with that small amount of sputum, which didn't bother him at all, and which he carelessly expectorated, he has killed them just as truly as if he had wilfully put poison

in their food. But such is often the case and such often are the results.

The "early" diagnosis of these cases would seem at present to rest chiefly with the sputum. This is unfortunate since really it means a relatively late diagnosis. In every case, however, the sputum should be examined daily, even for

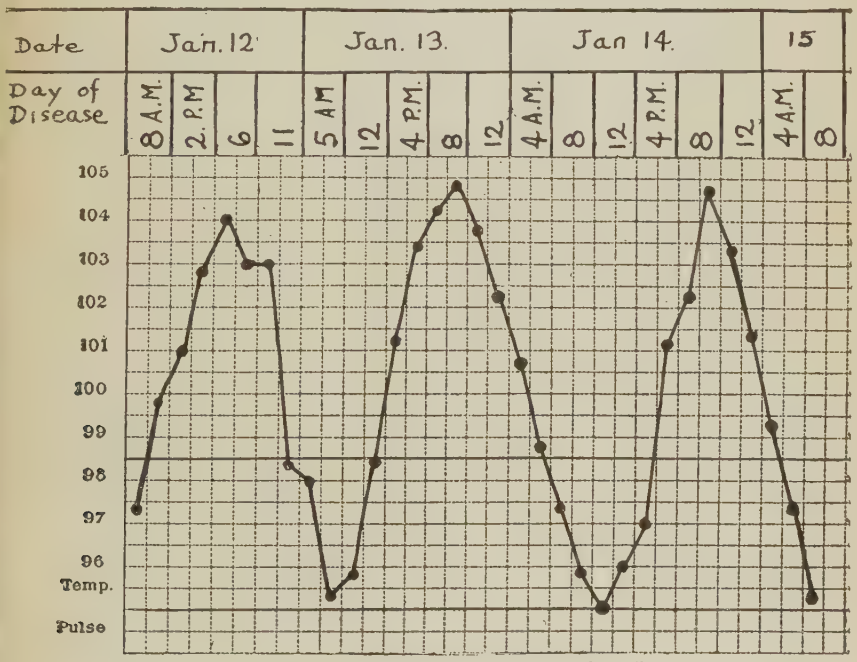


FIG. 143.—Chronic tuberculosis. "Hectic fever."

weeks, for a few tubercle bacilli may be found. The patient should be instructed to save the material which he raises when he clears his throat in the morning (page 431). The history of loss of weight, unusual fatigue each afternoon, and a slight daily fever between four and six o'clock in the afternoon, is suggestive but is not conclusive. The past history of a pleurisy or of a bloody sputum is of great importance, and the family history of relatives who had tuberculosis,

and the fact that the patient has lived or worked with one with this disease should be given considerable weight. X-ray plates of the chest are of value since at least they show whether or not the patient has in the past had much tuberculosis of the lungs. They nearly always show some "scars" and calcified glands which may (or may not) prove a past tuberculosis, but they seldom if ever decide the question whether or not this disease now is active. **The complement fixation test of the blood serum** has some value. As a matter of fact, the evidence of the disease can **best be found early by studying the family, the home, and the earlier history of this case,** and later by the physical examination of the lungs themselves; and yet all of the above methods should be used and as soon as the diagnosis of tuberculosis is made, active treatment should begin at once.

The tubercle bacillus, or Bacillus tuberculosis (Fig. 128), is one of the germs concerning which we know much. One judges it by its size and shape, but especially by the "fast colors" it assumes when stained with certain dyes and then treated with alcohol and acids that will bleach almost every other germ. Even experts, however, may be deceived. The sputum is often full of these bacilli; a consumptive able to work will daily expectorate from one to four billions of these germs. These bacilli are abundant in every place where men are expectorating carelessly. Why, then, are we not all consumptive? In the first place, because nearly all of these germs are dead when we inhale them, killed by that cheapest and best disinfectant, sunlight. In the second place, the soil (our bodies) into which these germs, seeds of disease, fall, must be a suitable one, else they are quickly killed by the protective agents in our tissues. We almost never inherit this disease, possibly we cannot inherit a soil which is favorable to its germ, but if the air in a house is always full of dust laden with these germs, what chance has the baby? For this reason tuberculosis is **more a house than a family disease.** The soil is made favorable or hostile by our manner of life also. These germs find a poor soil in the man

who lives an active, outdoor life with plenty of exercise, food and sleep. If, however, he has a **chronically infected nose or throat** he may contract it easily despite his outdoor life. The soil (our body) seems especially favorable for the germ **after an attack** of measles, whooping-cough, typhoid fever, small-pox, diabetes, and during any chronic disease, such as kidney or liver trouble. **An infected nose or tonsils** can keep the tuberculosis stirred up. In fact, the case may be truly stated as follows: we are all repeatedly exposed; the great majority of mankind have somewhere in the body before they die at least a small focus of this disease; about one third of all persons have at some time during their lives symptoms from this focus—perhaps a lung trouble or a pleurisy or enlarged glands or some bone or joint trouble; while one in every ten human beings dies of it. Of course every one must die sooner or later, but the tragedy of this disease is that, as a rule, it kills between the ages of 18 and 35—that is, takes those in the prime of life, and it makes so many young persons half invalids for years, and leaves so many of those who recover cripples and hunchbacks for the rest of their lives.

There are two varieties of tubercle bacilli which infect man: **the human form,** described above, and **the bovine form,** spread by the milk of tuberculous cattle, which infects especially the abdominal glands of children and very seldom the lungs.

Tuberculosis in its various forms kills about 100,000 a year in our country alone. And yet the saddest, as well as most hopeful, aspect of this whole problem is that were only ordinary rules of cleanliness observed our grandchildren would not know of the disease, except as a matter of history. To cure some diseases we may properly search for serums, antitoxins, and wonderful medicines, but to stamp out this disease, the worst of all, **will take only cleanliness.** The disease is caused by germs spread by a patient. Whether they reach the lung through the bronchial tree or we swallow them in milk and other foods is of little consequence; we get them from a person with tuberculosis, and he need not have

spread them. The germ gets into the body. It seldom causes any trouble at the point where it enters, but it is carried around in the blood or lymph stream and finds a spot where it can grow; in children in the lymph-nodes, bones or joints; in adults in the lungs especially, although any organ of our whole anatomy may be infected. Here it settles and here it multiplies, and here may be formed a focus of disease. But the germ also may remain quiescent for years in the spot where it settles, and then suddenly become active and cause symptoms.

Tubercle bacilli are short-lived in the open air and sunlight; in dark, damp rooms they may live for weeks. It is probably no exaggeration to say that if for two generations consumptives should cough into handkerchiefs and expectorate only into proper receptacles, and these should be properly cared for, the third generation would know the disease only by name. Probably the disease would rapidly disappear from cattle also, and thus this source of danger also would be removed.

The person with consumption need not be dangerous, but often is. A few cases in point are mentioned in Osler's text-book. In one physician's practice one chronic consumptive had buried four wives, one three and four two. Some day the laws will recognize such deaths as cases of homicide. These wives would not have died had their husbands been decently clean. The consumptive should train himself **to hold always a handkerchief before his face as he coughs.** He should always **expectorate into a suitable sputum cup**—one which can be burned with all its contents, boiled, or disinfected with strong fluids. He should remember that his sputum is poisonous and should treat it as such. The urine and stools also may contain many bacilli, but these are usually better cared for. He should not frequent dark rooms. It is no wonder that there is most tuberculosis among those who live in alleys and among the inmates of convents and prisons; in such places the death-rate from this disease may be 75 per cent instead of 10 per cent. A family moving into a

new house should always ascertain whether or not a consumptive lived there before them, and if one had, **the whole house should be properly cleaned, repapered, and repainted.** One case will illustrate this. A man in our employ died of chronic consumption. The health officials fumigated the room in which he died, officially oblivious of the fact that he had lived for months in the whole house. Then another employee moved into that house with his wife and four children, all in good health. In six months the wife and three children had died of consumption, later the baby died, and now the husband has a chronic cough. This is not an unusual case. Tuberculosis is **above all else a house disease,** and houses which have one case now will usually be found to have been the home of several cases in the past, or will become such in the future. The tubercle germs in the sputum, on the walls, or on the floor of a poorly lighted room can retain their virulence for six weeks or more. Who next will move into that house?

The first thing the patient with tuberculosis should do is **to have treated every focal infection which can be cured or removed** from his body: the tonsils, nasal sinuses, etc. This is important since these pyogenic infections certainly can keep the tuberculosis active. They explain the later flare-ups of lesions apparently cured. Persons who fear consumption should plan to work and live as much in the open air as possible, should sleep and eat as much as they can, should sleep with the windows wide open, should take a cold sponge every morning.

The prognosis of a case of tuberculosis will depend partly on the soil inherited. Some with advanced disease get fairly well, while those who have a bad family history must fight harder. Next, the outlook will depend on **how early the diagnosis was made.** Finally, the outlook will depend on the **vigor and conscientiousness with which the treatment is followed.** No matter how "early" the case, the patient should give up all work and for at least nine months should make the struggle for recovery his sole occupation.

The treatment is more a social question than a medical one. The doctor can make the diagnosis, but after that the cure rests with the patient, the nurse, and the friends. This treatment consists of **rest and of freedom from labor and, if possible, from worry.** Just so long as there is fever the patient should stay in bed. When there is no fever, and he is up, he should govern all his acts with reference to the treatment. There is no heroism in staying at work until one has to give up, but a foolish bravery, which too often results in death.

Fresh air and sunlight are the great curative measures. The air should be dry air if possible, at a high altitude, if convenient, but at least it should be fresh. While the patient is staying in bed, bed and all should be wheeled outdoors during the day, and during the night all the windows should be wide open, no matter how cold it is or how much rain or snow blows in. Clothed in proper flannels, a warm cap, and mittens, the patient soon enjoys such a life. When up, the patient must sit out or walk out in the open air, no matter what the weather. The patient is made to keep a record of the number of hours spent outdoors each day. The question often arises, "Is it necessary to go to a sanitarium?" And the answer is, "No, not necessary, although, if the patient can afford it, it is much better." The class work among the poor consumptives in our crowded cities has shown about as good results as have the most expensive sanitaria. There is some advantage in mountain air, but the chief advantage of a sanitarium is the discipline which the patient is made to feel and obey. It is seldom that consumptives willingly do at first what is best for them, since, unfortunately, these things are not the most comfortable. The rigorous routine of a well-organized institution does help in forming right habits, and the patient should stay until he has begun to enjoy those features of the treatment which were at first so disagreeable to him. In the city the small tent on the roof or in the back yard, in which the poor patient sleeps, is the salvation of many cases. The next point of importance is the food. **Of the**

foods most important, milk and eggs stand at the head of the list. We now do not emphasize the proteids of the diet as much as formerly since we recognize that an excess of these may lead to injury of the kidney. It has been found, however, that the more fat a patient eats the better are his chances of stemming the tide of the disease, for fat furnishes the blood with certain substances which inhibit others that bring about the breaking down of tissues. Indeed, it would appear that the chief advantage which cold climates offer for the care of patients with tuberculosis, in fact the chief advantage of open-air treatment in general, is that patients under these conditions naturally crave fats in much the same way that the lumberman of the North craves fat pork. We can now understand the partiality with which, a few years ago, doctors regarded cod-liver oil, a medicinal food which is now again coming into favor.

Eating is for consumptives often a hardship. In the majority of cases loss of appetite and dyspepsia are two frequent and early features of tuberculosis. The patient's appetite is "delicate"; he demands the "dainty" things, and family and friends sacrifice much to provide them. That is one sad feature of these cases. If not warned and advised the relatives will keep the patient in the nice, warm room which he enjoys so much, will shield him from every draught, will feed him with the most expensive things their means allow. We have but one remark to make about this condition. If the family were deliberately to plot against the consumptive's life and plan to put him out of existence as soon as possible, they could not, without resorting to poison or violence, choose a quicker way than that which we have described. Again, the patient demands to stay at home. He rebels at the hospital. He wishes to "die with his family," and he does so; or, more correctly, the whole family dies with or soon after him. Nature seems to have omitted only one precaution in this plan for the extermination of the unfit—she has not provided any way of burning down the house after the last member of the family dies.

Simple tonics are prescribed to improve the appetite, cold baths when the fever is high, some simple remedy to make the cough less distressing or to lessen the night sweats, some creosote perhaps, but to combat the disease there is as yet no drug, no serum, no "cure." The market is full of such remedies with their astonishing advertisements, but the actual result is the impoverishment of the patient, so far as his money goes, and the loss of valuable time which should have been spent in combating the disease.

Since this rational prophylaxis and treatment were begun the death-rate from tuberculosis in some cities is only one half what it was; thanks to the crusade against tuberculosis and other preventable diseases, our average age has been increased by over twelve years; now 50 per cent of those consumptives who take the treatment thoroughly become "arrested" cases and able to return to active life. The former opinion no longer prevails that each patient "goes the way of all consumptives to an early grave."

When a hæmorrhage occurs there is usually great excitement, and often many foolish things are done. If it is a small hæmorrhage, the patient should quickly be got into bed and kept as quiet as possible. He should lie on the diseased side if we know which it is. The foot of the bed should be elevated. For a day food is withheld, but cracked ice is allowed. Later, a light diet is given. Of drugs, opium, aconite, calcium lactate, and purges are the best; opium keeps the patient quiet and relieves the cough. Aconite, pituitary extract, and purgation reduce the blood pressure in the lungs, while calcium lactate increases the coagulability of the blood. The profuse hæmorrhages of opened arteries or ruptured miliary aneurisms in tuberculous cavities are often rapidly fatal, and all one can do in such a case is to keep the patient perfectly quiet for a few hours. If he faints, let him alone. This lowers the blood pressure, which is therefore beneficial. Opium is not given in cases of profuse hæmorrhage, since we wish the patient to cough the blood up.

Although the **cough** may be troublesome, it is necessary

in order to remove the sputum. Nevertheless since a very **irritating cough may be due to a tuberculous ulcer of the larynx,** the throat should be carefully examined and, if necessary, treated. **An irritating bronchial cough** can be relieved by inhalations (equal parts of creosote, turpentine, and benzoin; one teaspoonful in a pint of boiling water). A warm poultice on the chest will often give relief. In the morning a glass of hot water containing fifteen grains of bicarbonate of soda will often aid. **Medicines** to relieve the cough—cough syrup, etc.—usually contain opium and should be used only under the doctor's directions. The night sweats are less distressing when the patient uses flannel night clothes.

Tuberculous adenitis. **The lymph-nodes** are filters shaped like a kernel of wheat and scattered along lymph-vessels. It will be remembered that the lymph-vessels drain the fluid in which the body cells lie, and hence contain much of their ashes. They also drain away **the products of disease**—toxins and germs. The lymph-nodes filtering out retain these and thus protect the body. That is the reason why, when we have an infected finger, we are often sore in the armpit; the nodes in that place are fighting the germs. But often the nodes themselves are overcome in this struggle, and an abscess forms in them.

These nodes which are themselves the seat of tuberculosis swell often to the size of lima beans, and since they are in groups, may form masses even fist size. Sometimes the lymph-nodes win—kill off the germs in them—and slowly return to normal size. More often their centre becomes caseous, lime salts from the blood are deposited there, and the nodes remain throughout life a calcified mass. Often the nodes suppurate—that is, become an abscess, and the pus must find some outlet. While the local disease of these nodes is in itself not very important, yet so frequently are they the starting-point of an acute miliary tuberculosis that it is in fact a very important matter. Sometimes all the lymph-nodes of the body are affected, but this condition is rare. **The nodes most often involved in tuberculosis** are those of

the neck, of the root of the lungs, and of the mesentery of the intestine.

Tuberculosis of the nodes of the neck, which drain the mouth, nose and tonsils, is the most common form of this disease, especially in children. The popular names of this condition are "scrofula" and "kernels in the neck." This infection is aided by nasal catarrh, chronic throat catarrh, enlarged tonsils and adenoids. The tonsils themselves sometimes are tuberculous. Since there is good reason for believing that some cases of pulmonary consumption (others begin in the lung itself) are due to a chain of infection which includes the tonsils, the nodes of the neck, those of the root of the lung, and the apex of the lung,—the proper care of a child's nose, throat, and tonsils is strongly urged. For the most part the "kernels in the neck" disappear, though some may be left as hard, calcified lumps; others suppurate—then the bunch of lymph-nodes in the neck, which is composed of separate nodes, now mat together and become soft; an abscess "gathers" and breaks, leaving a sinus, or "running sore," which may remain open for months, and then close leaving the ugly scars so often seen in the neck.

The tracheobronchial lymph-nodes which drain the upper respiratory passages and the bronchial lymph-nodes which drain the lung are often infected in pulmonary tuberculosis, possibly before the lung. These cause few symptoms unless they get so large that they press on the trachea, causing paroxysms of coughing, or on the blood vessels, or on the nerves in the chest, or unless an abscess forms. But it is in these nodes that the disease smoulders for years, and from which it later spreads to other organs.

The nodes of the mesentery and those behind the peritoneal cavity drain the intestines, and since milk often contains tubercle bacilli (usually the bovine variety) these are frequently tuberculous, especially in young children. Sometimes these nodes are the chief lesion, forming large masses in the abdomen ("tabes mesenterica"), and producing a gradual loss of weight and strength, distension of the abdomen

and diarrhœa with thin, offensive, fatty stools. These symptoms are due chiefly to the disease, but partly to starvation since the fat, which is absorbed by the lymph-vessels now blocked by the diseased glands, is lost in the stools.

The treatment of tuberculosis of the lymph-nodes is the same as that of pulmonary consumption combined with X-ray treatment and it is remarkable how rapidly large glands will disappear. If they have suppurated, the abscess of course must be opened. But the careful removal of these glands by operation now is seldom done.

Acute miliary tuberculosis, which really is tuberculous septicæmia, is the worst form of this disease. The origin of these bacilli is the intima of the thoracic duct, which may be covered by ulcerating tubercles. From this focus they are poured into the blood stream, and so are carried throughout, starting up all over the body myriads of tiny "miliary" tubercles.

This is one of the **hardest fevers to diagnosticate,** unless it is known that the patient had previously had a chronic tuberculosis. Many a case is an almost exact clinical picture of typhoid fever, without, of course, the specific proof. Some cases start as cerebrospinal meningitis; some, as very severe bronchitis. The patient is very ill from the first, with high, irregular fever, and usually dies after a few days or weeks of illness. That many of these patients recover is shown on the X-ray plates by innumerable shadows of healed miliary tubercles scattered throughout both lungs of patients who now are quite well.

In some cases it is not the tuberculosis germs which are poured into the blood but the **tuberculous toxin** from some focus, usually lymph-nodes of the mediastinum and retroperitoneum. These patients recover. Some of these cases appear on our records as cases of "continued fever," or are mistaken for typhoid fever for "clinically" (without laboratory aid) the two diseases sometimes cannot be distinguished. Doubtless this explains the experience of those who declare that they have had typhoid fever more than once. If the localization

of this infection is chiefly in the lungs, the patients are cyanotic, their dyspnœa extreme, and their prostration profound.

Tuberculous meningitis, popularly known as "water on the brain," is common in children, although it also attacks adults, in which case it is usually more acute. It is a form of acute miliary tuberculosis with especial localization of the tubercles in the meninges.

The child with this disease has usually been rather ill for weeks; perhaps he is known to have a chronic tubercu-

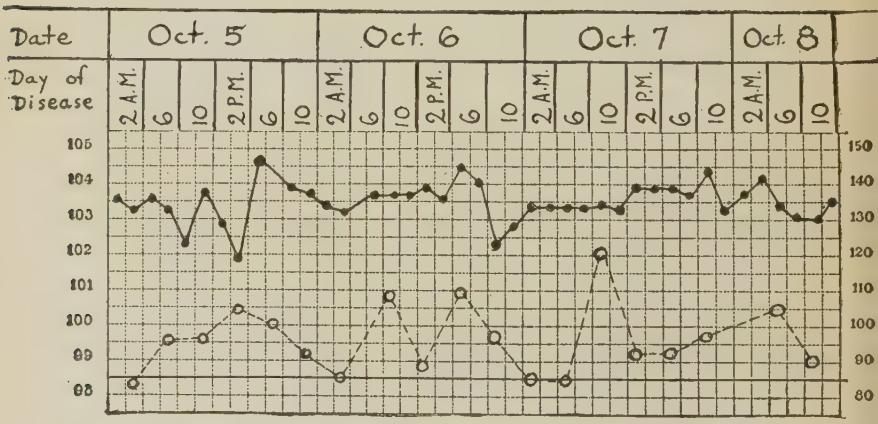


Fig. 144.—"Typhoidal fever" in a case of acute miliary tuberculosis.

losis; sometimes the meningitis follows measles or whooping-cough. **He gradually loses weight and strength;** has fever, then becomes very irritable, with headache and insomnia, and may show a complete change of disposition. **Then suddenly begins an acute illness** which in the initial stage, or **stage of irritation,** is marked by fever, vomiting, constipation, tympanites, headache, photophobia and sometimes by convulsions. **Stiffness of the neck** may be noted then. That is, attempt to raise the child's head from the pillow and one raises the whole body (orthotonos). **The cries of the child,** sometimes occasional and periodic, sometimes continuous, are very piercing (the "hydrocephalic cry") and indicate a terrible headache. **The pulse now is slow and irregular.** Gradu-

ally the cries cease, the child becomes dull, and hard to rouse. His emaciation is conspicuous. Finally the **stage of paralysis** begins, with paralysis of a few muscles (that of the eye is important) or of most of the body. The neck and back are often stiff, the head retracted. Convulsions, sometimes slight, sometimes severe, are not uncommon. The mental dulness deepens into coma, and finally the patient dies.

The diagnosis of tuberculous meningitis is made by lumbar puncture, as the tubercle bacillus can be found in the spinal fluid. Of almost equal importance with the discovery of the bacillus is the appearance after a few hours in the crystal-clear fluid of a **cobweb coagulum** which collects together to form the characteristic pellicle.

The outlook of this disease is practically hopeless. The treatment is to make the patient as comfortable as possible while conscious, and to keep up the nutrition by sufficient feeding. The lumbar puncture is sometimes made daily, as so many of the symptoms are due to the high pressure around the brain, and this is relieved by removing some of the fluid.

Tuberculosis of the serous membranes, that is of the pleural, pericardial, and peritoneal cavities, of young persons differs from other infections of these surfaces in that tuberculosis is so seldom limited to one cavity. A general rule is that infection of more than one cavity (polyserositis) of patients below middle life practically always means tuberculosis, of those older, cancer.

The two pleural cavities are not at all connected. In them the lungs lie free except at their hila where the bronchi and blood vessels enter. They are covered by the visceral pleura, which everywhere rubs against the parietal pleura, which covers the inner surface of the chest wall. Normally there is no cavity at all and everywhere two opposing surfaces are in contact and should rub with perfect freedom, for the movement between them during inspiration and expiration is considerable. But make a hole through the chest wall through which air can get between these two pleuræ, and the lung, like

a punctured balloon, will collapse to almost one sixth of its usual size. Then there is pleural "cavity" enough, and the condition is called **pneumothorax.** This may be caused by

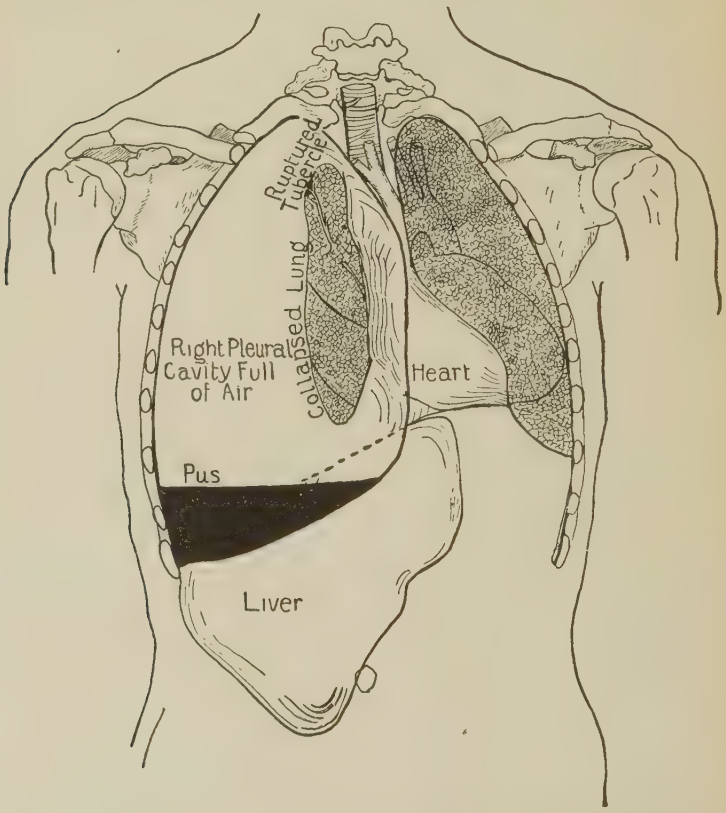


FIG. 145.—A diagram of the chest showing pneumothorax on the right side. A small tubercle on the surface of the right upper lobe has ruptured, allowing air to enter the pleural cavity from the bronchi. The elastic lung collapsed like a toy balloon which has been pierced. There is no longer the normal negative pressure in the right pleural cavity, but the pressure there is equal to or above the atmospheric pressure. The result is that the heart is pushed (or sucked by the negative pressure) to the left side and the liver falls. The contents of the ruptured tubercle will set up a pleurisy and pus will collect in the pleural cavity. The condition is pyopneumothorax.

a stab or a bullet which opens the pleural cavity from without; but in the vast majority of cases pneumothorax is due to a tubercle under the pleura, which by ulceration causes a

communication between a bronchus and the pleural cavity. (Fig. 145.)

The pericardium is the closed sac containing the heart, whose inner surfaces, like the heart's outer surface, is covered with a smooth, serous membrane. Thanks to these two membranes, there is no friction while with each beat the heart contracts and expands. Here also the word "cavity" is misleading, for there is no empty space between these two serous surfaces, until we open the outer surface and let air or fluid enter.

The abdominal cavity is everywhere lined with the peritoneum, a serous membrane, and all the organs within it—liver, stomach, bowels, and spleen—are covered with this membrane. These organs are in almost constant motion and should move freely without friction. On the other hand, the kidneys, pancreas, aorta, etc., lie behind and not in this cavity. The peritoneal cavity also is not a cavity until you open into it. It is true that the stomach and bowels are hollow organs with much empty space often filled with air, but the air in the stomach is not in the peritoneal cavity.

These serous surfaces of the pleura pericardium and pericardium are everywhere identical in structure and are affected by the same diseases, and yet so little communication have they that in only the two diseases mentioned above, tuberculosis and cancer, does infection of one reach the others.

Tuberculous polyserositis means tuberculous disease of any one or all of these serous sacs. It may be acute, subacute, or chronic. In acute inflammation tiny tubercles spring up over the serous membrane, spoiling its smooth, glossy surface; an exudate of bloody serum containing flakes of fibrin and pus cell exudes, and sticks the two pleural surfaces tightly together; then these fibrin "adhesions" become permanent—that is, are replaced by scar tissue—so that now the two surfaces which should rub together without friction are inseparably joined together—are "adherent." Thus the acute disease gets well, but leaves behind a chronic trouble—these adhesions. There is now at the point of the adhesions truly

no "cavity" left; it is entirely "obliterated." But sometimes in tuberculous serositis the exudate is so abundant that the serous sac is more or less filled by a large amount of fluid, clear, yellow blood serum usually, although when the trouble

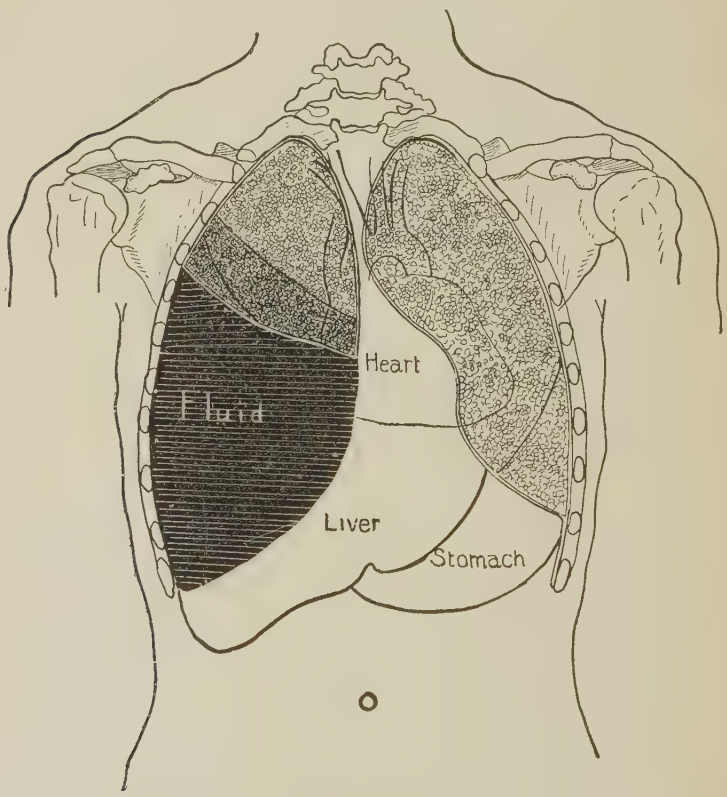


FIG. 146.—Diagram of the chest of a case with pleurisy with effusion on the right side. Note that the lung is above the fluid, and that the liver is lower than normal.

is very acute it is red because of red corpuscles. Such fluids clot readily, forming a jelly-like coagulum. When the opposing serous surfaces are not adherent, this fluid can distribute itself among the organs as gravity dictates. It makes room for itself in the abdomen by compressing some organs, as the stomach and bowels, by distending the abdominal walls

or pericardial sac, and in the chest by allowing the lungs to collapse. Soon this fluid is reabsorbed, and the serous surfaces may again become smooth, but often some adhesions are left.

In another and more chronic form of tuberculosis only a little fluid exudate collects, but **the serous membranes become thickened** by large conglomerate tubercles which may become cheesy. In a still more chronic form, there is no fluid exudate at all, but the serous membranes surrounding entire organs become thicker and thicker, because of a great growth of connective tissue in them, and finally instead of being about as thick as paper they are even a quarter of an inch thick, and this layer of scar tissue, by contracting, may greatly deform the organ which it encases.

The symptoms of tuberculous polyserositis will depend on the toxæmia of the general disease and on the local disturbances of the organs in the cavities involved.

Acute tuberculous pleurisy or fibrinous pleurisy begins usually with a chill, fever, a dry cough, and a "stitch in the side." Every motion of the lungs that makes the inflamed pleuræ rub over each other is attended by an exquisite pain, which cuts short every respiration. This disease usually lasts but a few hours or days. Healing, a few fibrous adhesions usually are left. An attack of fibrinous pleurisy is of itself but a trivial disease; but it is nevertheless a very important step in a tuberculous process, and the record of such an attack in the past history of a patient is now considered as good reason for suspecting that any later pulmonary trouble is tuberculosis. **The sharp pains of acute pleurisy** are relieved by strapping the chest with a tight binder or by broad strips of adhesive plaster, by local application of heat—the Paquelin cautery, the hot-water bottle, or a mustard plaster—by the ice-bag, by painting the chest with iodine, and, lastly, by morphia, which should be injected hypodermatically over the painful area in order to obtain, not only the general effect of this drug, but what is more important, the local anæsthesia of the skin area to which the pleural pain is referred.

Pleurisy with an effusion sometimes of three or four quarts of fluid in one or both pleural cavities is of itself a much more serious condition. This fluid is of higher specific gravity than that in a case of simple hydrothorax (e.g. in dropsy) and clots readily after it is removed. This condition if it develops spontaneously is always tuberculous in character. Proper examination of the fluid itself will usually reveal (in over 90 per cent of the cases) Bacillus tuberculosis. While pleurisy with effusion may begin acutely, with a chill, fever, and at first a fibrinous pleurisy, yet the majority of cases begin so insidiously that the date of onset cannot be stated. The patient not feeling well and losing weight and strength may continue at work until his condition is almost accidentally discovered. As the fluid in the pleural cavities, one or both, increases, the patient becomes more and more short of breath, since the collapsed lung can not "ventilate well," but it is remarkable how much fluid can accumulate, if it does so slowly, before the patient's symptoms suggest trouble in his chest. He is often treated first, and sometimes until death, for stomach trouble, cancer, or heart disease. This is a most calamitous mistake since pleurisy with effusion, if treated properly, is practically never fatal. There is always fever, often unsuspected, a little dry cough and a secondary anæmia. Of itself this disease also is usually merely an accident, but it is one step in the course of a more general tuberculosis. The majority of cases have no further pleural trouble after this one subsides, but others develop widespread pleural adhesions which later slowly contract and produce permanent deformities, drawing the chest wall in, the shoulder down, and the spine usually to the side. There are usually pains, sometimes severe, in the chest, and the patient is rather short of breath. Since his lung is more or less limited in its expansion it is abnormally susceptible to other diseases, especially pulmonary tuberculosis. This condition is called "chronic dry pleurisy," or "contracted chest," and its symptoms may continue throughout life.

The treatment of pleurisy with effusion is rest in bed, full

diet, cod-liver oil, and all the therapy of pulmonary tuberculosis. Formerly we tapped the chest repeatedly in order to keep it as empty of fluid as possible, but now aspiration is limited to those cases with dangerous amounts of fluid present.

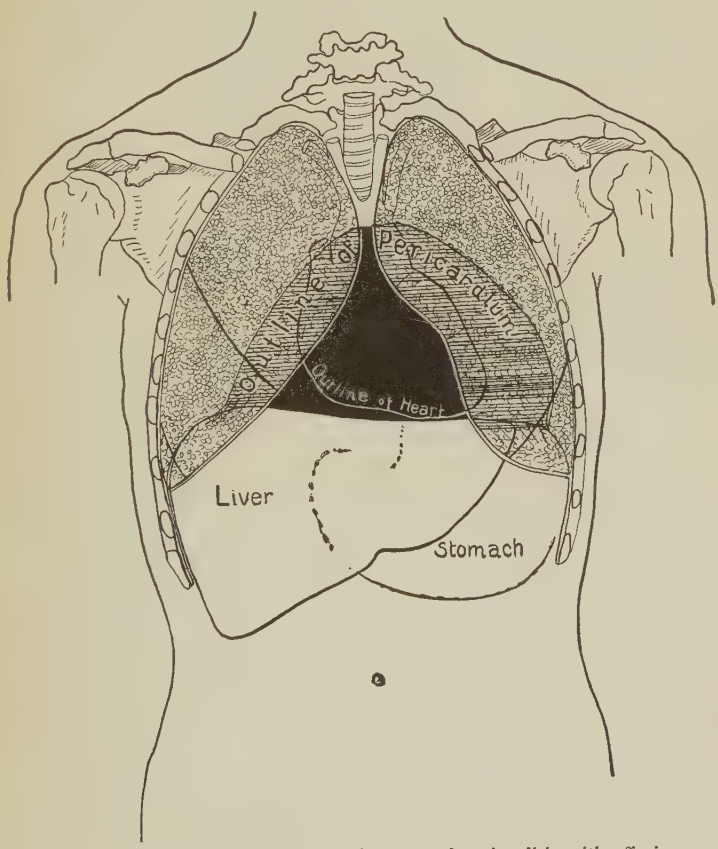


FIG. 147.—Diagram of the chest of a case of pericarditis with effusion.

Tuberculous pericarditis is one of the hardest conditions for even the doctor to recognize. When it is acute there is heard with each heartbeat a to-and-fro rubbing or scratching sound, which may be very loud, and which is increased by

the pressure of the stethoscope. Strange to say, this rub is usually painless, while the similar, but much fainter, pleural friction rub of fibrinous pleurisy is very painful (Fig. 138).

The presence of fluid in the pericardial cavity is hard to determine, because the case is usually mistaken for one of dilated heart; or, if much fluid is present we may think it is in the pleural cavity; and, indeed, if it collects slowly enough between one and two liters of bloody fluid may so distend the pericardial sac that this fills practically the whole left half of the chest. The symptoms which the nurse might detect are the following: increasing dyspnœa, but with the breathing free and painless; dusky cyanosis; a pulse that is weak,—especially so during each deep inspiration (in fact one or two beats during inspiration may entirely fail—"pulsus paradoxus"); and restless delirium. This fluid should, as soon as detected, be removed by tapping. The patient is kept very quiet, using morphia freely if necessary, an ice-bag is applied to the precordium, a diet prescribed which favors a low position of the diaphragm, and the bowels are kept as free as possible of gas.

Adhesive pericarditis, or adherent pericardium, is the result of an acute inflammation of that sac. The two layers of serous membrane become bound together, sometimes over the entire heart, by adhesions. Then at each heartbeat the pericardium must move with the heart. Since normally the outer surface of the pericardial sac is but loosely attached to surrounding organs, this condition if limited to the pericardial sac makes practically no trouble; but if the inflammation is severe enough to extend through this sac and to bind this to the surrounding organs, then the work of the heart is greatly increased and a very serious heart disease follows, yet one often difficult to recognize. The heart, which normally moves considerably during each contraction, now, in order to contract, must pull in the ribs, pull on the pleuræ and lungs, and pull up the diaphragm and along with it the liver, which is attached to the lower surface of that muscle. The result of the greatly increased work is a huge, dilated heart, and the

symptoms are those of marked valvular (relative) disease or "myocarditis."

Tuberculous peritonitis is very common, especially in the colored race and in older children and young adults. The infection is supposed to spread into the peritoneal cavity from caseous tuberculous lymph-nodes. The three forms of serous sac inflammation, acute, that with effusion, and the adhesive, mentioned in connection with pleurisy, occur here also, but the acute form usually produces so few symptoms that either the local condition is overlooked, or one of several wrong diagnoses (especially typhoid fever) is made. In general, however, we speak of a wet form and a dry form. The ascites, or effusion of free fluid in the peritoneal cavity, of the "wet" form may be extreme. On tapping these cases, which should be done, from ten to twenty liters may be removed and the fluid replaced by oxygen. There is usually present much tympanites (the distention of the bowels with gas), either diarrhœa or constipation, and an irregular fever. In the dry form a diagnosis of abdominal tumor often is made. This mistake is not a bad one, since often large tumors are present—masses of tuberculous matter walled in between the viscera, organs bound together, the omentum thickened and rolled up, or fluid exudates tightly encapsulated in various parts of the abdominal cavity. These cases all have more or less malaise, with loss of weight and strength, fever, and secondary anæmia.

Chronic adhesive tuberculous peritonitis produces remarkable anatomical disturbances of the abdominal organs and but few symptoms. The adhesions of scar tissue bind them together and greatly distort them. It may encase them in thick membranes. That the bowels are not more frequently obstructed is surprising. The treatment of tuberculous peritonitis is that of tuberculosis in general.

All cases of tuberculous peritonitis seem to do particularly well after **the simple operation of merely opening the abdomen,** exposing the bowels to the air, and closing the wound again. As a result the disease seems to disappear spon-

taneously. There now is considerable doubt, however, that this operation adds much to the therapy. The fact is that tuberculous peritonitis has a marked tendency to recover spontaneously, as is shown by the many cured cases discovered accidentally when later in life the abdomen is for any reason opened.

FIG. 148.—Kyphosis of spine (Pott's disease). (Indiana Univ. Dept. of Illustrations.)

Tuberculosis of bones and joints is one of the commonest forms of tuberculosis in children. Practically all cases of hunchback, "Pott's disease" (Fig. 139), of "hip disease," all "white swelling" of the joints, and of "cold abscess," are due to tuberculosis. The disease begins, as a rule, in the bone just bordering the joint and progresses slowly, causing great destruction of bone and of the articular surfaces, and leaves the joints stiff. The treatment is as complete rest for the joints as is possible, combined with that for tuberculosis in general.

Tuberculosis of the kidney is a quite common disease. The tubercle bacillus is usually carried to this organ by the blood, but in a few cases it travels up the ureter from the bladder (an "ascending infection"; tuberculosis of the lower genito-urinal tract is very common). The symptoms are those of pyelitis, that is the presence of pus, blood, and tubercle bacilli in the urine. Suggestive early symptoms are **an acid pyuria and a sudden hæmaturia.** The result is often the entire destruction of the kidney. The treatment is

the removal of the organ combined with the treatment for tuberculosis in general.

Tuberculosis of the adrenal glands, called "Addison's disease," deserves especial mention. The adrenals are two small bodies, one just above each kidney. If they are really glands, their secretion is an internal one—that is, it flows into the blood—for they have no duct. Just how these glands act normally we have no idea, but we know very well what happens when they fail in their function. Such patients **have attacks of exhaustion** (asthenia), which may come after exertion or spontaneously, during which they feel so weak that they hate even to turn in bed. There may be also pain in the back under the short ribs, but this is not always present. The pulse becomes so weak that it can scarcely be felt, and the systolic blood pressure surprisingly low (below 70 mm.). The person vomits incessantly without any relation to eating. At first these attacks occur at irregular intervals, but later the condition may be constant. At the same time, and usually this is the earliest symptom, the skin gets darker and darker, especially that of the face, hands, axillæ, nipples, waistline and groin, and dark spots appear in the mouth. The condition may progress for two or three years but always ends fatally.

Although this disease may be due to destruction of the adrenals from any cause, cancer for example, yet in the great majority of cases it is due to tuberculosis.

Some think that all of the symptoms of Addison's disease are due to loss of the internal secretion of the gland; others, that they are really due to extension of the disease from the glands to the abdominal nervous system covering them. In favor of the first view is the effect of the drug "adrenalin," or "epinephrin," which is an extract of these glands and is now widely used. When applied to a mucous membrane, for illustration, this drug causes marked local contraction of the blood vessels, and hence it is of great value in checking hæmorrhage. When injected into an animal it causes a marked rise of blood pressure. It may, when administered

hypodermatically to cases of Addison's disease, relieve the asthenia, but its effect is very transitory. One may try also arsenic and strychnia in large doses.

Tuberculin has proved of great value not only in the diagnosis of tuberculosis but also in its treatment. Tuberculin is the poison that the tubercle bacillus forms and sets free in the fluid in which it is growing. What we actually use is this liquid culture medium after the germs have been removed by filtration. If a very small dose (the initial dose is 0.0001 c.c. of old tuberculin, diluted to not more than 0.3 c.c. with normal salt solution) of this filtrate is injected intradermally, that is so superficially that the lumen of the needle is visible through the epithelium, at the end of from one to four days a red flat papule from 1.5 to 4 c.c. in diameter will rise at the seat of injection, and may persist for weeks. Such a reaction shows that there is in the body an active or an inactive tuberculous lesion. Such an injection into the skin of a normal person should produce no effect at all. We also may inject 0.0002 c.c. of the tuberculin subcutaneously and obtain a general systemic reaction, with headache, malaise, and during the next two days, usually in about eighteen hours, a rise of temperature. At the same time, and this is the value of the subcutaneous method, there is likely to be a "local reaction"—that is, for a few hours the tuberculous lesion giving the general test betrays its presence by flaring up. For illustration, if the tuberculous trouble is in the knee, that joint should become more painful, more acutely swollen, etc. Tuberculin is used also in the treatment of tuberculous patients without fever. Very small doses are injected subcutaneously, so small that they cause no fever, and at regular intervals. Some very good results are obtained in this way.

Epidemic Cerebrospinal Meningitis

Epidemic cerebrospinal meningitis also called "spotted fever," is due to a germ named Diplococcus intracellularis meningitidis, a very tiny diplococcus, the two cocci of the pair flattened against each other (see Fig. 127). Since it is so soon "swallowed" by leucocytes, it gets the name "intracellularis."

There have been several bad epidemics of this disease in the world, and so many in this country that some authorities consider it an "American disease." Large epidemics, however are rare. This disease often occurs sporadically, but more often in small epidemics, especially in crowded cities, in crowded institutions, in army camps, jails, etc., but also in country regions, where it attacks children especially. It seems not to be very contagious, since it is seldom that two persons in the same family, or house, have it.

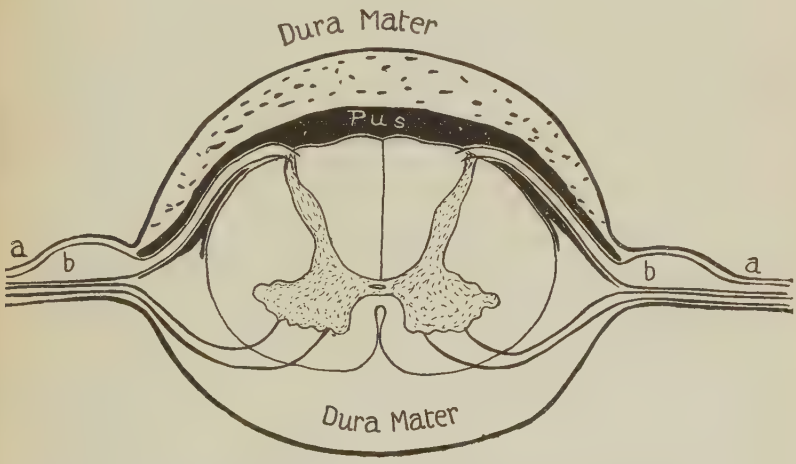


FIG. 149.—A cross-section of the spinal cord from a case of epidemic cerebro-spinal meningitis. Note that there is an increased amount of cerebro-spinal fluid, and that the pus is chiefly posterior. If a hollow needle is stuck through the dura a cloudy fluid will gush out. *a,* spinal nerve; *b,* posterior root ganglia.

While it reaches the body through the tonsils and nose, yet in about two thirds of the persons in whose nose this organism is abundant, it produces no disease. It is by examining their nasal secretions that we ascertain who, in a regiment of soldiers for illustration, these "meningitis carriers" are. Once inside the body, the germ is carried about in the blood stream, forming a true septicæmia which affects many organs, but which, as a rule, soon locates in the membranes which surround the brain.

Diplococcus intracellularis meningitidis attacks the pia

mater, the delicate membrane which immediately covers the brain and cord, and in which are the tiny vessels which provide the cortex with food. These blood vessels become congested and soon furnish an exudate of pus which covers especially the base of the brain and the posterior surface of the cord (Fig. 149). The inflammation spreads to the brain tissue itself and to the inner walls of its ventricles. In very acute cases, the patient dies from the toxin of the germ before the pus has had time to collect.

The symptoms of this disease are due chiefly to the direct action of this toxin on the cortex, to the pressure of the exudate around the brain and inside the ventricles, to the inflammation which spreads along the cranial nerves as they leave the brain, and, lastly, to the infection of other organs by this germ.

The disease may begin as a cold in the head or as a sinus attack, and often with joint and lung symptoms. Usually, however, **the onset is very sudden** with a chill, vomiting, headache, fever, stiffness of the neck, and drowsiness. The patient is in general dull and indifferent but at times irritable and restless, and, if disturbed, may be delirious, sometimes maniacal. Later comes stupor, and even coma. **The neck is stiff;** try to raise the head and you must lift the shoulders from the pillow. Sometimes the stiffness of the neck extends to the back, and the whole body is a rigid rod. **The headache** is excruciating and comes in spasms. There are exquisite **pains in the back and limbs.** Sometimes there are **convulsions.** The inflammation creeps along the optic nerve, and the patient at first **cannot endure any bright light**—"photophobia"—and later may be blind; along the auditory nerve, and he becomes very sensitive to even slight sounds, and later **becomes deaf;** along the motor nerves of the eyeball muscles and the patient becomes cross-eyed or wall-eyed; along the facial nerve, and there is facial paralysis, etc. **The fever** is usually very irregular. **Small subcutaneous hæmorrhages**—petechiæ—may appear under the skin, whence one name of the disease, "spotted fever." **Fever-blisters** are very common

on the lips. The increased intracerebral pressure is shown by the very slow pulse and the Cheyne-Stokes respiration, and can be demonstrated by lumbar puncture. There is usually a high leucocytosis.

During an epidemic some cases are scarcely ill; some are very ill and die in a few hours; more die within five days; but most of the cases are ill for weeks or months. Complication follows complication—pneumonia, arthritis, pleurisy, etc. The majority have some permanent sequel to remind them of this illness, as deafness, blindness, the paralysis of an eye muscle, or severe paroxysmal headaches, vomiting, convulsions, and mental feebleness.

The diagnosis is made by a lumbar puncture, for the cerebrospinal fluid, instead of being a very clear water, will look like thin milk and in it the germ can be found. The treatment early while the septicæmia is present is to give intravenously 100 c.c.s. of the **antimeningococcus serum** of Flexner, and later, after one is certain that the meningitis has begun, one removes by lumbar puncture about 40 c.c. of the spinal fluid and then replaces it with about 30 c.c. of this serum. This is repeated every day if necessary until the fever drops. This serum has lowered the mortality of some epidemics from over 80 per cent to less than 30 per cent.

The patient's strength must be kept up by diet and stimulation, and he be made comfortable in a dark room or in a warm bath or with an ice bag at his head.

Diphtheria

That diphtheria is caused by Bacillus diphtheriæ (Fig. 128) there is no doubt. This germ lodges usually on the mucous membrane of the throat, multiplies there rapidly, kills the surface of this membrane, and causes an inflammation with the exudation of much fibrin. The epithelial surface of the dead mucous membrane and the fibrin forms **a white, leathery skin, or false membrane,** which gives the name "diphtheria" (leather). Strip off this membrane and the bleeding surface of the submucous tissue is exposed. The germ forms a virulent toxin which is absorbed into the blood and carried

over the body. This toxin causes the fever, pains, and all the other general symptoms, in fact the entire disease except the membrane. The germs themselves all remain in the membrane; only in the severest cases do they invade the blood stream. They are on the body, not in it.

The onset of symptoms in a case of diphtheria occurs usually from two to five days after the exposure. It begins with fever, chilly feelings, pains in the back and limbs, headache, and general malaise. At once the patient notices that his throat is slightly sore and a **little patch of white membrane** is seen, usually on a tonsil. The throat does not look very red, nor are the tonsils much swollen. The glands in the neck swell, and the face becomes ashen gray. The next day one notices that the patch of membrane has enlarged and has crept beyond the tonsil. Then one is sure of the diagnosis. This membrane may **grow rapidly,** extending over the soft palate to the posterior wall of the pharynx, up into the nose, far down to the larynx and trachea, and even into the fine bronchi. It may extend along the Eustachian tube to the middle ear, along the nose into the nasal sinuses, and down the œsophagus into the stomach. The membrane when small in extent is white, thin, and delicate, but yellow and tough when it is thick. The tissue below it may be very necrotic and slough deeply. In the ordinary case the fever is never high and soon falls to normal. The throat is not very sore, and the patient doesn't feel very ill—not nearly so ill as in acute tonsillitis. This is really unfortunate, for diphtheria is a far more serious illness. After about ten days the membrane in untreated cases with recovery loosens, and falls off in shreds.

Bacteriological examination is necessary for diagnosis, since some cases really cannot be differentiated on inspection alone from acute tonsillitis, and other cases have no membrane at all. **A culture is easily taken.** The State Boards of Health provide at suitable points boxes containing two tubes, the one containing a sterile swab, the other a tube of Lœfflers' serum (glucose bouillon and blood serum). The nurse care-

fully removes the swab from the tube and, not allowing it to touch elsewhere, she rubs it against the membrane in the throat. She then, using the technic of the bacteriological laboratory, rubs this cotton on the media and sends the tube at once to the laboratory. If this tube is examined between eighteen and twenty-four hours after the culture is made the diagnosis of diphtheria is easy. After twenty-four hours, however, other germs will have overgrown this germ. Again, other germs, especially streptococci, can cause a membrane which to the inexperienced resembles that of diphtheria.

Some patients are terribly ill from the extreme toxæmia of diphtheria. Other cases at no time feel ill, and can only with difficulty be kept quiet. From the amount of membrane one cannot judge the severity of the case; some fatal cases have little membrane, others much. The severest cases have no fever, and even a subnormal temperature until death.

Virulent diphtheria bacilli live on for weeks in the throats and noses of many whose illness has entirely recovered. These, and the mild cases of nasal diphtheria without visible membrane, are very dangerous to others, for while they may have no symptoms yet for months they may spread the disease to others (carriers).

One of the worst forms of diphtheria in children spreads to the **larynx and is called membranous croup.** This child may have a rough, croupy cough for a day or two, then suddenly almost suffocates. He tosses about, sits up in bed, and struggles to draw his breath. He becomes quite blue. Severe cases, if not relieved by tracheotomy or intubation, will suffocate. In milder cases the paroxysms are soon over but may recur later.

Bacillus diphtheriæ can cause, in addition to diphtheria of the mucous membranes, a diphtheria of the skin around recent wounds and diphtheria of the eye, which is a serious condition.

Diphtheria is a disease always present (i.e. endemic) in the community, but severe epidemics sometimes spread, especially in country regions. Not only is it spread by mild cases,

convalescents, and carriers, but also by particles of the membrane clinging to clothes, etc. **The germ has wonderful endurance** and can live in clothes or toys, etc., for even five months. It is also carried in milk, in which it multiplies without souring it or in any other appreciable way altering it. It attacks children for the most part, although adults—nurses and doctors especially—are often affected.

Among the serious complications of diphtheria are the following: acute circulatory failure (often called toxic myocarditis although the term is misleading) the common cause of sudden death of diphtheria patients; severe nephritis, due to the diphtheria toxin; and bronchopneumonia, due to an extension of the inflammation from throat to lung, a common cause of death. Many persons tell of coughing up "tubes" from the lungs. These tubes are the lining of diphtheritic membrane from the larger bronchi. Streptococcus is an organism which seems to flourish in company with Bacillus diphtheriæ, and causes many of the severe inflammations in diphtheria cases.

Among the important sequelæ of diphtheria are various paralyses, especially of the throat and eye muscles, and, less often, of the limbs, which often (in almost one fifth of the cases) develop during the second and third weeks of convalescence. The diphtheria itself may have been so slight that it was not even suspected until the paralysis of the throat begins, when the voice becomes nasal, and the patient begins to regurgitate liquids through his nose.

About 70 per cent of all children **are immune to diphtheria** and so will not catch it when exposed. It is, therefore, of great importance to find out which children are not immune and therefore might catch it. This can be done by means of the **Schick test** which is made is follows: A small amount of diphtheria toxin (one fiftieth of a minimal lethal dose for the guinea pig in 0.1 c.c. of normal salt solution) is injected into (not under) the skin of the forearm about two inches below the bend of the elbow. If the child is not immune, that is, if the test is positive, a red and slightly raised spot

(from one to two centimeters in diameter) will appear in from twenty-four to forty-eight hours at the point of injection, will reach its maximum size in four days, will persist for from seven to ten days and then fade leaving a superficial scaling and a persistent brownish pigmentation. A control test is made by treating the other arm in exactly the same way, only using a toxin which had previously been heated for ten minutes at 75° C. A positive test means that the child's blood contains no antitoxin and therefore that he is susceptible and should be made immune by means of **three injections,** at one week intervals, of the **toxin-antitoxin mixture.**

Since the use of **diphtheria antitoxin** has become general the dread of diphtheria has to a large degree disappeared. Formerly it was a very serious disease, which killed over half of those whom it attacked; but during the past few years, thanks to this therapy, the mortality has fallen to 10 per cent. Could the use of antitoxin be still more universal, this mortality would drop even more. This enormous saving of children's lives, of whole families even, can scarcely be appreciated.

A germ is dangerous because of the poison, that is toxin, that it produces. The diphtheria bacillus, for illustration, scarcely ever reaches the tissues of the body. It first forms the membrane on the wall of the throat, and in this it multiplies and produces its toxin, which soaks into the blood and produces every one of the general symptoms of the disease. At once, however, the body begins to produce an antidote for this toxin, a specific antitoxin which when sufficiently potent cures the patient. But it happens in about half the cases that the toxin kills the patient before his body has produced sufficient antitoxin. Now it makes little difference whence this antitoxin comes. The antitoxin from another person or from an animal that has recovered from diphtheria if we inject it into him will do as well for our patient as that antitoxin produced by his own body. So the method is as follows: A horse is made ill by injecting into him a dose of

diphtheria toxin. The bacilli are not injected, for accurate dosage would be impossible if we used live organisms, as these could multiply and kill the animal. But fortunately the bacilli pour their toxin into the fluid in which they are growing, and by filtering this fluid free from bacilli we get its toxin and so can use it in accurate doses. The first dose is one estimated as being just short of a fatal dose. The animal becomes very ill, but recovers, by manufacturing antitoxin enough to neutralize the dose given. Give a second dose of the same size and no illness would follow; but double the dose and the horse will be as ill as before, and, when it recovers, its blood will contain an antitoxin potent enough to handle two of the original doses. The next time three doses of toxin must be given, then four, then five, and so on until the animal is able to stand a thousand or more of the original doses without being ill. Its blood-serum will then contain antitoxin of great potency, and a little of this injected into our patient's body would greatly help out by supplying "ready made" a large quantity of the same antitoxin that his own body is trying to manufacture. Then the horse is bled, and his serum put in little bottles, one dose in each. On every bottle is a label stating the number of "units" (500, 1000, 3000, etc.), that its contents contain. What "500 units" means we have not space enough to explain, but it refers to the potency of that particular specimen of antitoxin, when measured by its effect on guinea pigs.

Diphtheria antitoxin may be given subcutaneously, or, if the case is critical, intravenously. No time should be lost. Whenever diphtheria is even suspected, the antitoxin should be given at once, even before a sure diagnosis has been made. One big dose is better than several smaller ones. In an ordinary case we inject about 6000 units of antitoxin at once and repeat the injection at intervals until we see the membrane shrivel up and loosen. If there is a membrane on the larynx, 16,000 units are injected at one dose. If the child, even a tiny baby, is very ill, even 10,000 units may be given in the course of a day. The antitoxin itself does very little harm,

although sudden deaths have followed its use, but these were probably cases of status lymphaticus which should not have been given an antitoxin. **Serum sickness,** however, is common (it occurs in about 10 per cent of all cases treated) especially in persons who have been subject to hay-fever asthma or urticaria (hives). Serum sickness may begin half an hour after the injection, or even eight to fifteen days later. Its symptoms are fever, a skin rash, swelling and pain in the joints, and œdema. Persons who have ever had hives, asthma or hay-fever **should be tested first by injecting intradermally** 0.025 c.c. of the antitoxin diluted 1:100 with sterile salt solution. If in ten minutes an urticarial wheal rises at the point of injection then the antitoxin (concentrated) should be injected in 0.1 c.c. lots each twenty minutes. An easier, but less safe, way is to inject 0.5 c.c. first and to give the full dose four hours later.

A child with diphtheria should be nursed in a cool, well-ventilated room. If there is difficulty in breathing, the air should be saturated with moisture, which can be supplied by a steam kettle designed for that purpose. The patient must rest quietly in bed, even into the convalescence, and the **heart should be guarded against sudden movements,** since circulatory collapse is an all too frequent cause of death in diphtheria. Liquid diet is given, and as much water as is safe considering the condition of the heart. If a child with laryngeal diphtheria is in danger of suffocation, he should either be **intubated or tracheotomy performed.** If the former, a tube for the child to breathe through is put into the larynx. Then the problem of feeding, which begins on the third day later, becomes important since the head of the child when fed should be a little lower than the body. Tracheotomy is the operation of opening the trachea just below the larynx and inserting a tracheotomy tube. The nurse must later watch the tube that its two tapes are always in place, must keep a piece of gauze over the shield, must frequently remove the inner tube in order to keep it clean, and if necessary should feed the child through a nasal tube.

The local treatment of the throat should be thorough and regular. Swabs, gargles, etc., are given regularly. When giving them the nurse must be very careful that the child does not cough some of the membrane into her face. Those exposed to diphtheria should themselves take an occasional dose of antitoxin, perhaps 1000 units, **as a prophylactic measure.** All persons whose throats or noses harbor the diphtheria bacillus should be isolated, no matter how slight or how longstanding their infections, for they spread the disease.

Acute Dysentery

Shiga's bacillus, or rather Shiga's group of bacilli, causes much of the **acute dysentery of infants and adults.** It occurs especially in the Tropics, is very contagious and may cause terrible epidemics; it is the scourge of armies. While there are no real ulcers in the bowels, yet in a severe case large areas of mucosa are necrotic; indeed, the entire colon wall may be practically killed by the toxin of these bacilli.

The onset is usually sudden, with fever, abdominal pain, and the passage of small amounts of blood, mucus, and pus. There is constant desire to defecate, and great straining during the attempt. The severity of the symptoms, the prostration, and the toxæmia increase, and death may occur in a few days. The milder cases are self-limiting, with recovery in eight or nine days. Others last two or three weeks, and the subacute cases even months.

We are sure that this bacillus is spread by drinking water polluted by the stools of infected persons, some of whom are sick, and others, bacillus carriers, themselves well. Therefore the same quarantine precautions should be enforced as for typhoid fever.

The diet should be milk and broth. Should curds appear in the stools other liquids should be substituted for milk. **Rectal irrigations** are the best treatment, given with the patient lying with his hips elevated on a pillow, using water at 100°, which contains alum, or lead acetate, or, best of all, silver nitrate. The rectum is very sensitive and irritable,

and must be anæsthetized before the irrigation by a cocaine suppository. Many drugs are used,—Epsom salts, laudanum, ipecacuanha, corrosive sublimate, and bismuth. Some serums seem of value. Morphia is the only drug that will quiet the pain and tenesmus.

CHAPTER XIX

The Acute Exanthemata

Under the general title **acute exanthemata** are grouped a few very contagious acute fevers, especially of childhood, all with a skin rash as an important feature.

Scarlet Fever

Scarlet fever is an acute infectious and a very contagious (not as contagious as measles, however, since only a few of those exposed "catch" it) disease caused by Streptococcus scarlatinæ, and characterized by acute fever, a scarlet skin rash, and a sore throat. It is a disease especially of the first eight years of life and **is by far most fatal during the first two years.** Scarlet fever occurs in epidemics, chiefly in the autumn and winter—that is during the school months. The portal of entry of the germ is the pharynx, although it may enter through any fresh wound of the body. It usually is spread through direct contact by the nasal and mouth secretions (less by the desquamating skin), and is communicated after its acute onset, not before. The virus, however, **can live for months** in garments, toys, etc., and may be spread by contaminated milk.

The rise in temperature of scarlet fever begins in from one to seven (usually in three or four) days after exposure. **The onset** is very sudden, in most cases with vomiting, and often, in children, with a convulsion. The temperature rises during the first day to 104° or 105° F. The face becomes flushed, but with a peculiar pallor around the mouth and nose. The skin is dry and hot, the tongue furred, and the throat sore. On the second day, sometimes on the first, **the vivid scarlet colored skin rash appears.** This consists of tiny red dots which surround the hair follicles, and which rest on a flushed surface. It appears on the neck and chest, then spreads rapidly, covering the whole trunk in about twenty-four hours. It affects the face least and sometimes not at all. This rash is not a "breaking out" but an intense conges-

tion or "erythema" of the skin which disappears on pressure, and which therefore disappears with death, when the blood leaves the skin. The skin is swollen and tense and often itches intensely. The most characteristic rash of scarlet fever, however, is the crop of **fine red dots which appears early on the soft palate and roof of the mouth.**

The tongue at first is heavily coated along its centre, its edges vividly red, and its papillæ characteristically swollen and projecting through the heavy white fur, giving it the name **strawberry tongue.** This is often enough for diagnosis.

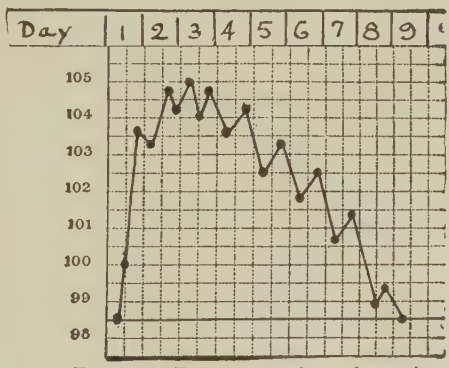


FIG. 150.—Temperature chart of a typical case of scarlet fever (v. Strümpell's curve redrawn from Sahli).

It is still more characteristic, however, after the desquamation of its fur, when it is smooth, dark red, and dotted with swollen papillæ (the "raspberry tongue").

The throat is always sore. Sometimes it is merely red and swollen, but in severe cases it is covered with a membrane which strongly resembles that of severe diphtheria.

The spleen is much enlarged and there is high leucocytosis.

The other symptoms are those of any fever.

After two or three days the fever begins to fall, the rash to fade, and is gone in about a week, and the skin, which now is dry and rough, begins to peel in fine scales and in large

sheets. **This desquamation** continues till the whole cuticle is shed, which takes from eight to about fifty days. The process apparently begins on the neck and chest, but the very first traces of it are seen under the finger nails, a very early sign of the disease.

No acute infectious fever varies so markedly in severity. Some of its epidemics are the mildest in character, some are very severe. Some of the mild cases have no rash at all; others have a rash so slight that it is easily overlooked. The claim is made that in some school epidemics 20 per cent of the cases **have no scarlet rash and no fever.** These are the ones who spread the disease. Other cases are recognized only after some sequela, as nephritis, has developed. **The most malignant (the fulminant) cases may** die in from twenty-four to thirty-six hours after the first symptom. Such cases have a temperature even as high as 108° to 109° F., convulsions and coma. **The hæmorrhagic cases** die in two or three days after the onset and are marked by extensive hæmorrhages which may cover almost the whole skin. They bleed also from the nose, mouth, kidneys, etc. In other cases the throat symptoms are so severe that a diagnosis of "malignant diphtheria" is made.

Few diseases **have more complications and sequelæ** than has scarlet fever. Among them are **acute otitis media** with rapid necrosis of the temporal bone, one of the most common causes of deafness; **nephritis of all grades** of severity, one of its most common and serious complications, which develops in from 10 per cent to 20 per cent of all cases, and also one of the most important sequelæ, developing during convalescence, sometimes as late as the fourth week after the fever is over, and the starting-point for very many of those cases of Bright's disease that seem to begin later in adult life; severe arthritis, a common complication which usually is a streptococcus infection of the joints; enlarged lymph-nodes; streptococcus endocarditis and pericarditis; etc.

Scarlet fever, when well developed, **is seldom mistaken for any other malady.** The cases which escape detection,

however, are those with little or no scarlet rash and with slight or no fever. **Measles** is sometimes confused with it, but a child with measles usually has felt miserable for several days before the rash appears, and the rash is quite different in character. **Diphtheria** is a much more difficult disease to exclude unless the diphtheria bacillus itself is found, since it may cause a similar rash, while on the other hand scarlet fever may cause a membrane in the throat which suggests diphtheria. Some patients with septicæmia, and patients sensitive to the administration of belladonna, quinine, potassium iodide, or diphtheria antitoxin, may have a rash which closely resembles that of scarlet fever. **The Dick test promises much assistance** in detecting the children who if exposed would be likely to catch this disease. These should be immunized by the subcutaneous injection of scarlet fever streptococcus toxin.

The mortality of scarlet fever varies greatly, depending both on the community infected and on the epidemic. In general it varies from 5 per cent to 30 per cent, but a fair average is 10 per cent.

At the first sign of the disease the patient should receive a full therapeutic dose of the antitoxin. Isolation should begin promptly. Fortunately the disease is not very contagious in its early stages. **The treatment is simple,** consisting chiefly of good nursing. The hospital is the proper and, so far as the community is concerned, the safest place for the child with scarlet fever. If the child is to be cared for at home the patient and nurse (or mother) should be rigidly quarantined from the rest of the family. The child should be nursed in a large, cool room from which all carpets, rugs and hangings have been removed. He should wear a flannel gown. A well fitting ice-bag or cold compresses should be applied around the neck early to control the cervical adenitis. Later, if these glands get sore or soft, hot compresses are better. Older children should gargle a mild antiseptic solution each three or four hours. The nose should be kept clean and crusts prevented by the frequent insertion of 0.5 per cent

menthol or liquid albolene. The ear should be examined daily (even when there is no pain) and if its drum is at all infected the external canal filled with a plug of cotton saturated with 7 per cent carbolated glycerin. Coma and convulsions are met by venesection (250 c.c. of blood is removed in case of children, and 500 in adults) followed by the introduction of an equal volume of normal salt solution.

The best diet for a case of scarlet fever consists of liquids, chiefly milk and fruit juices, and large quantities of water. It must be admitted, however, that the cases on pure milk diet do no better than those on a freer diet. **The bowels** should be freely moved, and cold sponge baths should be given when the patient is delirious or the fever high. To prevent complications the child should be kept in bed for at least three weeks, better still a month, after the temperature has become continuously normal. **The urine should be carefully examined daily** for signs of nephritis. The throat may need swabbing and the ear-drums, puncturing. **When desquamation begins** the patient should be rubbed almost daily with liquid vaseline and bathed every second day with soap. After four weeks of normal temperature and no complications the child may sit up an hour a day, and then for increased periods daily. **The quarantine** should continue until all desquamation is over and also until all discharges from nose and ears have ceased. Caffeine (strong coffee) is a good heart stimulant.

Acute exfoliating dermatitis is a rare and very interesting disease, which may explain the "second attacks of scarlet fever." It is a fever of sudden onset, with a course of five or six days, a scarlatinal rash, and marked desquamation, but without throat or tongue features, and not contagious.

Measles

Measles is an acute fever which manifests itself chiefly in the upper respiratory tract, and by a characteristic rash. Its germ is not yet definitely known, although some think it is a streptococcus. One reason why so few children escape is

that the infective agent is contained in the secretions of the eyes, nose and mouth, and is most communicable for several days before the rash has appeared. It is much less, perhaps not at all, contagious after the rash is well out.

The first symptoms appear in from seven to eighteen days, usually about fourteen, after the child has been exposed. **The catarrhal stage,** which lasts from three to four days, begins as a cold in the head, with some fever and malaise. The patient feels wretched. Diagnosis is almost impossible at the very first of the prodromal period and yet it is during

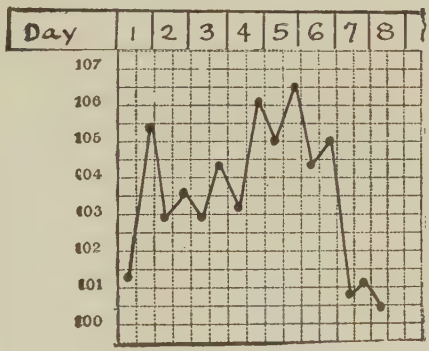


Fig. 151.—Temperature chart of a typical case of measles. (v. Strümpell's chart redrawn from Sahli.)

this stage that the disease is very contagious. **Koplik's spots,** little blue dots, pin point in size, each with a red areola, and most numerous on the inner side of the cheek opposite the first molar teeth, appear a day or two later, and are an early and very sure sign of the disease. These spots are seen in 97 per cent of all patients, and usually one to three days before the skin rash appears. Soon begin headache, nausea (perhaps vomiting), and chilly feelings. **The coryza** increases, as is indicated by coughing, sneezing, and redness of the eyes and lids, and marked photophobia. As the temperature rises the skin of the face especially is flushed and feels hot and tingling. The tongue is furred, and the mucous membrane of the mouth and throat is very red.

After about four days of the catarrhal stage the **skin rash appears,** first on the forehead, then on the face, and then in less than three days over the whole body. It begins as little red spots, "like flea bites," which, as they increase in number, become arranged in groups, sometimes of crescentic shape. These dots are little papules, very slightly raised above the skin, and can just be felt. Sometimes they are palpable enough to be confused with those of smallpox.

On the fifth or sixth day of the fever the symptoms begin to abate, and a fine, branny desquamation of the skin begins which is complete in some cases in a few days, in others, in several weeks.

The spleen is not enlarged, and a leukopenia is present.

In very severe cases, usually fatal, the rash, instead of little red papules which disappear on pressure **consists of minute hemorrhages** under the skin (black measles). The patients thus affected may bleed from the mouth, the bowel, etc.

The complications of measles are important but not immediately serious. Among these are severe nosebleed; laryngitis; ear troubles (otitis media) which follow measles more often than scarlet fever but the post-measles cases are milder; severe bronchitis and, especially, bronchopneumonia, a very common and dangerous complication which explains most of the fatal cases of measles especially of infants and the aged; bad inflammations of the mouth; Bright's disease (often); heart trouble (seldom); severe arthritis; sometimes paralysis, meningitis, and brain abscess. Some cases are left with a chronic coryza and enlarged tonsils and adenoids. Tuberculosis is very apt to follow measles.

The diagnosis of measles is very difficult before the rash is out, and yet it is then that the disease is especially contagious. Fortunately **Koplik's spots appear early.** The slow onset excludes scarlet fever.

The mortality from measles varies from 2 per cent to 10 per cent in most countries, but when introduced into a population which previously has never known it—that is, "into

a virgin soil"—the disease is terribly fatal. In the valley of the Amazon, for illustration, it is feared more than is smallpox.

The disease is spread by all the secretions including those of the mouth, throat, and nose. The poison clings to the clothes, linen, toys, furniture, etc., and may be carried by a third person. It will survive for a long time in the patient's room, unless this is properly fumigated. Measles is a hard disease to eliminate once it breaks out in a hospital.

The patient should be kept quiet in bed as long as there is any fever or any complication. The air in his room should be fresh and cool, but great care should be taken to keep him from catching cold, for bronchopneumonia is much to be feared as a complication, and tuberculosis as a sequela of measles. The diet should be simple, with abundant fluids, and the bowels kept loose by simple laxatives. All bright light should be avoided, since it may cause intense distress. The eyes should be kept clean with drops of saline or boric acid solution, the edges of the lids kept moist with vaseline, and the nose with albolene. The catarrh of the head and throat is relieved by inhalations of compound tincture of benzoin and by camphorated oil rubs over the chest. Hot drinks will help bring out the rash. If the fever is high, cold sponges are indicated. Cough syrups or paregoric will quiet the cough. During desquamation oil rubs are useful.

German Measles

German measles is an acute fever best described as "having the rash of measles and the throat of scarlet fever," but it really has no relation to either disease. Its most distinctive features are **its long period of incubation, its short invasive stage** (twenty-four hours of pharyngitis), its **benign course,** and its almost **absence of complication.** It is very contagious, but is usually very mild. Direct contact apparently is necessary for transmission. The period of incubation is from ten to twenty-two days. At the onset there are slight fever, headache, pain in the back and limbs, coryza, and a

characteristic adenitis of the mastoid, occipital, and cervical glands.

The skin rash appears on the first or second day, at first on the face, and in twenty-four hours has spread over the whole body. It consists of little pink raised spots arranged in patches. After two or three days it fades, leaving no stain. Koplik's spots are never present.

This fever differs from measles in that there are fewer prodromal symptoms, there is little or no fever, the rash is more diffuse, it is a little brighter in color, and its patches are less crescentic in shape than those of measles. Complications are practically unknown. Quarantine should continue for, at least, ten days. The treatment is that of any fever.

Smallpox

Smallpox, or variola vera, once the most dreaded of diseases, now is so seldom seen that it is little feared. It is an acute fever with a rash which cannot be mistaken.

The first symptoms appear in from five to twenty-one (usually twelve) days after the patient has contracted the disease. The onset is sudden, often accompanied by a chill, and, in children, by a convulsion. Then appear three symptoms, which, when they occur together, should always lead one to suspect smallpox: **intense headache, intense pain in the back and limbs, and vomiting.** The temperature quickly rises to 104° F. or higher, the pulse becomes rapid, and a restless delirium is very common. An early transitory rash, similar either to that of scarlet fever or of measles, may confuse one.

On the fourth day after this onset the true smallpox rash develops. **Small bright red spots** ("macules") appear on the forehead and wrists, and, in a few hours later, on the face, limbs, and trunk. These macules **soon become papules,** that is, they become raised and feel like shot in the skin. With their appearance the temperature falls and the patient feels better. On the fifth and sixth day of the disease—that is, on the second and third after the appearance of these macules, these papules **develop into vesicles,** by the development on

each of a cap of fairly clear fluid. This cap is not always dome-shaped (as in chickenpox) but more often is depressed in the centre, that is, it is "umbilicated." The fluid of these vesicles is never quite as clear a serum as in chickenpox and soon becomes full of leucocytes. **Then the pustular stage** has begun. These pustules have a characteristic greenish or grayish yellow color, and are surrounded each by a narrow areola of inflamed skin. The transformation of vesicles to pustules begins on the face and is complete over the body by the eighth day of the illness.

With the pustule formation the **secondary fever** of the disease, the "fever of suppuration," begins. The temperature, which for one or two days had been quite or almost normal, rises again, and the general symptoms return. This stage may in a mild case last only about twenty-four hours, but it usually is longer. By the tenth or eleventh day of the disease the fever again is gone and convalescence begins. The pustules dry down to "crusts" which, beginning on the face on the fourteenth or fifteenth day of the disease, gradually drop off, the most of them leaving a scar, or "pit."

The spleen is enlarged. The leucocyte count earlier was normal with the mononuclears increased, but during the pustular stage there is a moderate leucocytosis.

Such is smallpox, a disease with an incubation period of five to twenty-one (usually eight to twelve), with an initial fever of four days, a pause, and then the secondary fever; and with a skin eruption which passes successively through practically all the stages and forms of skin lesions—macule, papule, vesicle, pustule, scab, and scar. The rash is always most abundant on the face, hands, and feet; somewhat less abundant on the arms and legs; and, in even very severe cases, scanty over the trunk. It is present also in the mouth and throat. A point of great importance in diagnosis is that at any one time all of the skin lesions are of practically the same age—that is, one finds only papules, or only pustules, etc. This skin rash has a peculiar odor which is unmistakable. One can detect it not only in the sick-room but also

in closed carriages which just before them had contained patients, and because of it doctors have picked out smallpox patients in a crowd.

The case we described above was one of **discrete** smallpox, that is, one the separate pustules of which are scattered. **In the confluent type,** however, pustules on the face and hands are so numerous and crowded that they coalesce to form large superficial abscesses covering the whole face and head, which may be frightfully swollen, the features not recognizable. This is one of the most horrible and terrifying pictures we ever see in the sick-room. Such a patient, if he recovers, will be badly pitted.

Confluent cases are much more severe than the discrete. In them the rash often appears a little before the fourth day, and the fever of suppuration lasts much longer than in a discrete case. There usually is delirium. The death in fatal confluent cases occurs usually on about the tenth or eleventh day. In cases with recovery, the crusts may not all have separated even in a month.

Hæmorrhagic smallpox is a very virulent type of this disease. In its mildest form, one finds **little hæmorrhages into the vesicles** of a severe case of the confluent type but the skin in the true hæmorrhagic form, "black smallpox," consists of **subcutaneous and subconjunctival hæmorrhages** which appear on the second or third day. The patient presents a frightful picture, his skin of a deep purple color, the eyes bloody, the face swollen. He bleeds from the mouth, nose, lungs, rectus, kidneys. While a few recover the most die early, even on the third day, before the real smallpox rash has had time to appear.

Smallpox formerly was one of the most terrible of epidemic diseases. Practically no one escaped. Its mortality varied much, but for the most it was from 25 per cent to 35 per cent. No age was immune. The young especially suffered, since over 80 per cent of the fatal cases were children under ten years of age. Vaccination has robbed this disease of almost all its terror and now epidemics can occur only in an

unvaccinated community. Mild cases, however, are common, sometimes, among the unvaccinated, sometimes among those once vaccinated. We find them in the out-patient departments, cases of the discrete type at the height of the attack, but who complain only of a past headache or backache, and who now feel fairly comfortable, or who come with some minor complaint unrelated to their smallpox. Such cases are often passed over as chickenpox, "Cuban itch," etc. Because they are so mild, these are the dangerous cases, for it is they who spread the disease, and those to whom they give it may develop the most virulent form.

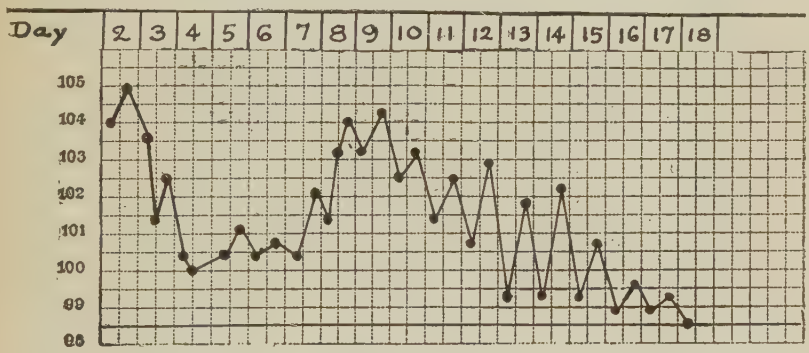


FIG. 152.—Temperature chart of a typical case of smallpox. (v. Strümpell's curve redrawn from Sahli.)

The complications of smallpox (laryngitis with œdema of the larynx, bronchitis, bronchopneumonia and nephritis) are most apt to develop early in the stage of eruption when the patient feels so well that he is careless.

The cause of smallpox is not surely known, but evidence points to a tiny parasite found in the skin. It is the dust of the skin that seems especially dangerous, and this clings with great tenacity to clothes, furniture, buildings, etc.

Every nurse should be alert to detect an early case of smallpox. Any patient acutely ill for less than four days, with severe headache, backache, and vomiting should be an object of suspicion, and doubly so if he can show no recent vaccination scar. Any person with even a few papules or

vesicles or pustules or crusts on his forehead and wrists is suspicious, provided these developed recently, at about the same time, and all are in about the same stage. It makes no difference how comfortable the patient may then feel. Chickenpox does occur in adults but so rarely that every case should be detained until seen by a physician and pronounced "not smallpox" before he is allowed to go.

The treatment of smallpox is chiefly a matter of nursing. The patient must be removed to a suitable hospital. The severe pains of onset may be relieved by phenacetin (10 gr.), acetylsalicylate (10 grs.), or morphia. One prescribes the usual treatment for fevers; hydrotherapy, even a continuous bath in confluent cases; and liquid diet, determined much by the condition of the urine. Since the eruption is less severe if the skin is protected from bright light, the room is kept dark. During the vesicular and pustular stages the affected parts are kept covered with lint masks soaked in ice-water containing some mild antiseptic (dilute mercuric bichloride, for instance), and protected with oil-cloth. Later the crusts are kept moist with vaseline. There is no way to prevent some pitting, but since the pitting is worse if the pustules and scabs are scratched or picked, it is **important to relieve the itching** to the best of our ability or the restlessness and scratching it causes will disturb the crusts and increase the pitting. To do this one uses calomine zinc lotion, phenol (two per cent), or even morphia. Paregoric will check the diarrhœa. The eyes must be kept washed out with boric solution, or 20 per cent argyrol, otherwise the patient may lose his sight. He should be kept in bed until disquamation is complete and should be held in isolation until every scab has separated.

One of the greatest triumphs of medical science is the discovery that **vaccination with cowpox** is a protection against smallpox. This has transformed a wide-spread virulent epidemic disease with a mortality of about 30 per cent, to a rare disease with a mortality of from 6 per cent to 8 per cent. An old and common custom was to inoculate healthy persons

with the pus from a case of smallpox. The result was a case of true smallpox, but one which as a rule was mild (though sometimes fatal), and which was just as contagious as the severest, doubtless more so, since the patient did not stay in bed. Since Jenner's day healthy persons have been vaccinated with lymph from a calf, with "cowpox" ("animal lymph"), or from the vaccination pustules of another person ("humanized lymph"). The disease produced by vaccination, "vaccinia," is not contagious and is scarcely ever serious. Cowpox is a fever of the cow, with pustular eruptions on the udder. The contents of these pustules are used in vaccinating calves, and from their pustules is obtained the lymph which is used for men.

The spot on the arm or leg to be vaccinated is first **scrubbed thoroughly with soap and water.** The soap is then rinsed off with sterile water. This is sufficient preparation. Some prefer to clean the skin with antiseptics, as for a surgical operation. If this is done, however, the antiseptic should be washed off thoroughly with sterile water and then the skin, well protected by a sterile dressing, allowed several hours in which to recover since this preparation seems to lessen the chance of a "take." To vaccinate, the superficial layers of epidermis are **scratched a little with a needle,** a knife, or an ivory point, or the upper layers of skin cut with a knife. The object is to expose the deeper epithelial cells but not to draw any blood. **The lymph is rubbed in** with the instrument used in scratching, allowed to dry, and the spot then kept covered for a few hours by a dry sterilized gauze sponge held on with adhesive. **Shields** are of doubtful value. Bunion plasters should never be used. They have caused tetanus.

On the **third day after vaccination a papule** appears, surrounded by a red zone. By the fifth or sixth this has become an umbilicated vesicle which continues to increase in size until the eighth day. The clear contents of the vesicles soon change to pus, and on the tenth day we have a fully developed pustule surrounded by an areola of swollen, tender, red skin.

The arm and the glands in the armpit are often very sore. **Deep scars covered by shiny skin** and with thick margins are the result of **secondary pus infections.** On the eleventh and twelfth days the areola disappears, and by the fourteenth the pustule has dried to a brown scab. During the following week this gradually separates and falls off, leaving a scar, which in its best form is **quite superficial and its base covered with small pin-point-like holes.**

An attack of vaccinia will protect the patient from smallpox for ten or fifteen years, at the end of which time he should again be vaccinated.

If the vaccination follows any other than the above described course, the result must be **considered doubtful.** If one is not careful, the spot vaccinated may become infected with pus organisms. There is no danger of inoculating any other disease into the system, as tuberculosis, lues, lockjaw, etc., if one uses ordinary care, an animal serum, and avoids dirty dressings, as a bunion plaster.

Infants are vaccinated after their second or third month of life. All persons exposed to smallpox should at **once be revaccinated.** Vaccinated persons may contract smallpox, but the attack, as a rule, is very mild ("varioloid").

Chickenpox

Chickenpox (varicella) is an acute contagious disease of unknown origin characterized by an eruption of vesicles. **The period of incubation** is from ten to fifteen (four to twenty-seven) days. **The onset** often is with a chill, vomiting, and pain in the back. Children, as a rule, do not feel ill, but some adults have constitutional symptoms severer than those of mild smallpox. **The rash** appears within twenty-four hours from the onset of the fever, first as red papules, then vesicles full of clear fluid, which are superficial, dome shaped, and which usually have no areola of inflamed skin. After two days this clear fluid has become pus, and two days later these pustules will have dried, forming dark-brown crusts, which later fall off leaving, as a rule, no scar, although they may if the patient has scratched them. Since successive

crops of vesicles appear each two or three days, all stages of the rash are present at the same time.

The vesicles of chickenpox appear first on the trunk, back, and chest; seldom on the face. Some may appear in the mouth. They vary in number from a dozen or more to several hundreds. They never coalesce. Complications are rare, and yet gangrene of, and hæmorrhages into, the pustules have been described, while cases complicated by nephritis and paralysis have been reported.

The early **differential diagnosis between chickenpox and smallpox** usually is easy, since the vesicles of chickenpox appear earlier in the disease, are translucent blisters, without a greenish or grayish yellow color, which rest very superficially on the skin, which are not umbilicated, and which have no areolæ. They appear first on the trunk, and later over the body in successive crops, so that soon all stages of vesicles and pustules are present at the same time. The vesicles of smallpox, on the contrary, are not superficial but rest on inflamed papules, appear first on the face, and are never really translucent. Indeed, to find even one vesicle containing a really clear serum is sufficient for a diagnosis of chickenpox. After the first few days, however, it must sometimes be difficult to make a correct diagnosis, since several recent severe epidemics of smallpox followed the mistake of diagnosticating the first cases seen as varicella.

Chickenpox is an extremely contagious disease, especially among children.

The treatment is simple, since the disease is mild and self limiting. The chief problem is to protect others, which is difficult because children who feel well object to six weeks of complete isolation. A very few cases have proved fatal, but there is doubt as to the correctness of the diagnosis. All possible precautions to control the itching are used (see page 482) since scratching may leave scars.

CHAPTER XX

Other Communicable Diseases

Mumps

Mumps is a very common contagious disease, due to a filterable virus, which, although a general infection, tends to localize in the salivary glands, especially the parotids (whence the scientific name **epidemic parotitis**). It affects, however, various other organs also, especially those of the genital, nervous, digestive, circulatory, and endocrine systems. Children between five and fifteen years of age especially are susceptible. Mumps occur in **epidemic form** in schools, armies, etc. **Transmission** is through the **saliva,** and its contagiousness is greatest at the very beginning of the attack.

Mumps begins eighteen or twenty days (from 8 to 30 days) after the child has been exposed, with fever, malaise, and often a sharp pain on swallowing something sour. A day or two later appears the swelling of one or more of the salivary glands, sometimes a submaxillary or a sublingual gland, but more often one of the parotids. The position of this early swelling of a parotid gland is very important in diagnosis. It begins just below and in front of the lobule of the ear, which it lifts just a little. Swollen lymph-nodes, a mistake in diagnosis often made, are situated a little lower in the neck and do not lift the lobule.

The swelling of the involved salivary gland increases for about two days, during which time usually other salivary glands also become involved, although their involvement may be delayed even six weeks. The skin over the gland is never red. At the height of the attack the mouth can scarcely be opened, there is pain on swallowing, and often earache. The fever is high and the prostration profound. After seven or more days the fever and the swelling gradually subside. While some children with mumps are scarcely ill, others, and especially adults, are very sick. Mild cases last

but four to seven days; severe ones two or three weeks. **Orchitis** in boys, oophoritis in girls, is a common complication. One, several, or all of the salivary glands may be involved. The idea that if some of the glands escape in one attack they may be involved in a later attack of mumps is not correct.

The patients, especially those beyond childhood, should be **kept quiet in bed,** not only until the temperature is normal, but until all glandular swellings have entirely subsided. This, though difficult, is the only safe course, since serious complications—orchitis etc.,—may develop. Cases complicated by pneumonia, œdema of the glottis, nephritis, and meningo-encephalitis may prove fatal. The subcutaneous injection of convalescent serum during the first week after exposure to mumps may, it is claimed, prevent an attack in an exposed child.

The treatment is rest in bed and liquid diet. Hot or cold compresses or belladonna ointment may be applied to the swellings. The mouth should be kept very clean. Cold sponge baths are very agreeable while the fever is high. The **quarantine** should be continued for at least two weeks from the onset in the mildest cases, and in severer cases until all symptoms have disappeared.

There are several other forms of **parotitis** which are not contagious, such as that due to lues, that which complicates typhoid fever, pneumonia, etc., and that following operation.

Should the gland suppurate, it must of course be opened.

Whooping Cough

Whooping cough or pertussis is an acute contagious bronchitis due to Bacillus pertussis, which occurs in all seasons, and sometimes, especially in the winter and early spring, in definite epidemics.

The period of incubation is about ten days. The first or **catarrhal stage** of this disease begins like an ordinary cold in the head, with slight fever, running eyes, and a cough of increasing severity. As a rule no suspicion of the nature of the disease is aroused during this first week, although the

expert may notice that the cough is unusually insistent. Following the catarrhal stage begins the **paroxysmal stage,** which dates from the first whoop; then the diagnosis is no longer in doubt. Other paroxysms of coughing are brief, with an inspiration after each two or three coughs, but in this disease the child attempts the impossible feat of coughing fifteen or twenty times on one single expiration. Of course in the attempt the lungs become forcibly compressed by the strong muscles of expiration, and at last fill again with one long inspiration, which it draws with a whoop. **During these paroxysms** the face becomes blue, its veins swell, the eyes bulge out, their whites are injected, and the child looks as though he might suffocate. The result of such a paroxysm of coughing may be the expectoration of a **little mass of very sticky mucus,** in which the specific germs can easily be found. A child may have very few such paroxysms or even a hundred a day. They are induced by swallowing, by any irritation in the throat, and by the emotions. Some children vomit at the end of each paroxysm and if these are very numerous they may almost starve. This stage lasts from three to four weeks, during which the paroxysms become less and less frequent.

The complications of whooping cough are many. During the paroxysms the strain on the lungs, **heart,** and blood vessels is terrible. Blood vessels may burst, and the child bleed from his nose, eyes, ears, or lungs; or the bleeding may be under the skin, or into the brain with paralysis as the result. The lungs are often injured; sometimes they become emphysematous. Sometimes they literally burst and the air makes its way along the bronchi and windpipe and appears under the skin of the neck.

The worst **complication** of whooping cough is **bronchopneumonia.** Because of it whooping cough is one of the most fatal of the acute infectious diseases for children under five years of age, and a very serious disease for the aged.

The treatment of whooping cough is, first of all, to isolate

the child rigidly and for a long time. While this disease is probably most contagious during its catarrhal stage—that is, during the week before a positive diagnosis is made—yet it often continues to be contagious after the acute illness is over, provided catarrhal symptoms persist, and the quarantine should be continued until these all have disappeared.

During the paroxysmal stage the child should be kept in bed and if possible in the open air. All influences which tend to precipitate the paroxysms should be avoided. Various protective **vaccines** are used and often with good results. **X-ray therapy** of the chest sometimes helps, probably be-

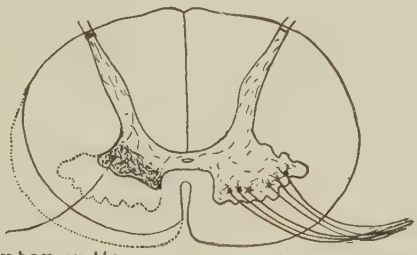


FIG. 153.—Cross-section of the cord of a case of anterior poliomyelitis. Note that the right horn has atrophied, its cells have disappeared, and that the anterior root of the spinal nerve is shrunken.

cause it favors the resolution of the enlarged tracheobronchial nodes. The severe cases with much vomiting **should be fed frequently** and regularly and in addition given a little food after each paroxysm. Of drugs, **paregoric** should be freely allowed.

The convalescence of whooping cough is tedious, and watchful care should be taken that the child does not catch cold or overdo. It is just at that time that bronchopneumonia as a complication usually develops, hence it is at just this period that the greatest care is necessary, especially in the case of infants. **The chronic cough** which sometimes persists for months after an attack of whooping cough is best treated with X-ray exposure of the mediastinum, cod-liver oil, fresh air, a nourishing diet, and, if possible, a change of climate.

Anterior Poliomyelitis

Anterior poliomyelitis, the cause of the common form of **infantile paralysis,** is an acute infectious disease which begins as a general infection, but which usually causes also scattered lesions throughout the central nervous system, especially in the anterior horns of the spinal cord (hence the name, "anterior," "polio," or gray, and "myelitis," or inflammation of the spinal cord). In these infected areas of the anterior horns the motor nerve cells die, and, consequently, their fibres also die. If a nerve fibre is injured a new one will grow out from the cell, but if the cell itself dies it is never restored, and the muscle which its nerve fibre supplies will die also.

Anterior poliomyelitis attacks especially infants during the second year of life, frequently children between the ages of three and seven, and sometimes, but rarely, adults. It is caused by a germ spread apparently by mild cases who themselves develop no paralysis. Mothers of the affected children, however, usually unjustly blame the nurses for "letting the child fall" or for "letting it catch cold."

The disease begins acutely with a cold in the head or with gastrointestinal symptoms. The fever lasts for one or two days, during which the child becomes restless or somnolent and always irritable. In a few hours the mother notices that one or more of his limbs is not moved normally. The paralysis shows none of the "system" or "order" common to other paralyses. Sometimes the paralysis is extensive; sometimes it affects only a few muscles of one or more limbs, sometimes, only parts of one or more muscles. At the end of the first day the paralysis is at its height, and would seem to be extensive; but a gradual improvement begins at once, since the cells injured but not destroyed soon regain their function, but those actually destroyed, never. The result is that whole muscles, or portions of muscles, atrophy, that is, waste away. Later, if all the muscles of a limb are paralyzed, this limb, as the child grows, fails to develop normally and remains throughout life shorter and smaller than its mate.

Since the lesion is one of the lower motor neuron the affected limb is weak, limp, and flail-like. If a muscle is affected and its antagonist is not, the latter will pull unopposed, and so will bend the joint and may cause a serious deformity. This is the way in which many club-feet, etc., arise. There is seldom any disturbance to the sensations of the limb, since, as a rule, the motor cells only are affected.

The diagnosis is easy. If a child or adult has an arm, leg, or foot shorter, smaller, and weaker than the other, and the joints of this limb are loose, that is, are flail-like so that this limb dangles and is of little use, the chances are that the condition is due to this disease. If it dates from infancy and is "due to a fall," there is practically no doubt as to the diagnosis.

The treatment of anterior poliomyelitis is very unsatisfactory. If during the very early stages of an attack one could inject into the exposed child the serum of a convalescent case (15 c.c.s. intraspinally and 100 c.c. intravenously) we might ward off the paralysis. After the paralysis has begun the child should at first be let alone. A few days later, the muscles not entirely paralyzed will get much stronger if daily they are carefully massaged and exercised by electricity. This must be done systematically. After twelve months there is no benefit from treatment. Many of the later, distressing deformities can be corrected by a surgeon, who will transplant the whole or part of the tendon of a healthy muscle so that it takes the place of a useless muscle.

Erysipelas

Erysipelas is an acute, diffuse, spreading inflammation of the skin caused by Streptococcus hæmolyticus (Fig. 127) and characterized by the scarlet color of the affected area and the absence of pus formation. Now, it sometimes occurs in epidemics, especially in the spring, but in olden times it repeatedly spread through the wards of hospitals, making such institutions very dangerous places for the sick. It really is not a very easily communicated disease and so is not always quarantined in medical wards, but it is very quickly con-

tracted by persons with fresh skin wounds, therefore by post operative cases, by women just after labor, and by newborn infants because of their freshly cut cord. A severe case of erysipelas does not always remain a skin infection but becomes a septicæmia causing abscesses in the internal organs and a fatal endocarditis.

The period of incubation of facial erysipelas lasts from one to three days, after which begins fever, often accompanied by a chill, and a flush appears, first on the bridge of the nose, then over both cheeks, giving the "butterfly rash," and which sometimes spreads over the head, neck, chest and even over almost the entire body. The area of involved skin becomes red, hot, smooth, swollen, and œdematous, and has a well-defined raised edge. Blebs often form on the inflamed skin, and, in severe cases, small abscesses. Swollen lids may close the eyes; the lips may become huge. After four or five days of high continued fever the temperature falls often by crisis. Among previously healthy persons the mortality from erysipelas is only from 4 to 7 per cent., but among drunkards and old persons it is a serious disease.

Erysipelas is a "self-limiting" disease, whose course cannot be shortened. One gives drugs to give comfort, good tonics, and applies to the skin cloths wet with cold water saturated with magnesium sulphate, or weak antiseptics, or, instead of water, one may use ichthyol. The patient should lie quietly, receiving a nutritious diet, and considerable fluids.

Influenza

Influenza, or la grippe, is a very contagious epidemic, even pandemic, disease, which more than once has swept with rapidity over practically the whole civilized world, prostrating even 40 per cent. of all exposed. Between epidemics we often see sporadic cases of this disease, although many of these cases are acute exacerbations of a sinusitis which a previous unrecognized or mild attack of influenza had caused.

The germ of influenza has not been identified, although the last organism to claim consideration is Bacterium pneu-

mosintes, a minute, filterable, bacillus-like organism. For years the specific organism was supposed to be Bacillus influenzæ (Fig. 128), the germ most commonly found in simple and in complicated cases of grippe. Later, several other germs also were blamed, including streptococci and pneumococci. Now, however, these are considered as secondary invaders which cause the various complications of influenza: pneumonia, empyema, etc.

True influenza has several very **characteristic features:** suddenness of onset with fever, profound prostration, severe aching pains in the back and extremities, conjunctival injection, a rapidly progressive inflammation of the respiratory mucous membrane, and leucopenia.

The disease may, and in the largest group of cases does, begin as an acute sinusitis, bronchitis, pleurisy, or bronchopneumonia, but with a more abrupt onset and with far more prostration than is common in these conditions. In a second large group of cases the nervous features predominate—headache, profound depression, prostration, etc. In still another group the gastro-intestinal tract is especially affected, and the patient suffers from nausea, vomiting, abdominal pain, diarrhœa, jaundice, collapse, etc. Finally, and this is a very important group of cases, the disease causes no local signs, but the influenza may, with almost typical chills, simulate malaria, or may, when the fever is continuous, simulate typhoid fever.

In general the acute fever of influenza lasts only from one to four days. Then, as a rule, follow one or more of the very numerous serious complications and sequelæ of this disease, due probably to organisms for which the influenza germ seems to have prepared the soil; as pericarditis, endocarditis, septicæmia, peritonitis, appendicitis, nephritis, chronic bronchitis, bronchiectasis, and often otitis media. It may cause nervous troubles of almost all descriptions—meningitis, abscess of the brain, paralysis, and melancholia; it may even be followed by dementia.

That influenza itself seldom kills is indicated by its imme-

diate mortality of only 0.5 per cent. Its complications, however, especially pneumonia and empyema, are exceedingly fatal. As a disease which destroys the health of those who recover from its acute attacks few can equal it. The continued ill health which follows it, however is usually due to continuing infections of the nasal sinuses, tonsils, gall-bladder, appendix, bronchial tree, etc., which the acute disease leaves behind, and some of which may be relieved by operation.

The diagnosis of influenza during an epidemic is easy, but that of a sporadic case is more difficult. The extreme nervous exhaustion it causes is very suggestive.

The treatment of influenza is, first of all, isolation, and the disinfection of the secretions, especially the sputum, although this may prove to be unnecessary, since, strange to say, the disease would seem to be contagious only before its acute onset. The patient must be kept in bed from the first until convalescence is well established, since this is the best way to prevent complications and sequelæ. He should receive from 3000 to 5000 c.c.s. of fluid daily, the fullest diet possible, and should be well purged and stimulated.

The convalescence is long and tedious; it may take months, it may take even years before the patient is well. For this reason a change of climate, when possible, is a great aid. Of course there are many vaccines on sale for both the prevention and treatment of influenza, but none have as yet proven satisfactory. A transfusion, using as donor one who recently has recovered from this disease, may save life.

Epidemic Encephalitis

Epidemic encephalitis, encephalitis lethargica, the "sleeping disease," is an epidemic disease which accompanied the terrible influenza epidemic of 1919. The cases of sleeping sickness were at first supposed by some to be cases of influenza with special involvement of the nervous system, and by others, to be cases of botulism. Soon, however, this form of encephalitis was recognized as a distinct epidemic disease.

The period of its **incubation** may be about two weeks. **The onset** of the lethargic or somnolent-ophthalmoplegic type is

usually slow and insidious, often with fever, which lasts but a few days, restlessness, insomnia and double vision. Soon, however, (in 80 per cent of the cases), lethargy appears.

The lethargy of this encephalitis varies from slight mental inertia to even complete unconsciousness. The severe cases usually can be aroused from their stupor sufficiently to answer questions. Those less severe sit or lie quietly, the face smooth and expressionless, the eye muscles often, and sometimes the facial muscles also, paralyzed. They scarcely move unless spoken to, and then reply very slowly. Put a good meal before a marked case and, although underfed, he may merely look at it for an hour. Usually, that he may eat it the food must be put into his mouth. And yet, ask such a patient a question and to your surprise his answer, although slow, may show a keen mind. They look as though mentally greatly reduced and yet they insist that the political or social news in the papers be read to them. In milder cases a very suggestive symptom is incontinence of urine and feces in a patient who mentally would seem fairly alert.

The sequelæ of epidemic encephalitis are very important; insomnia, various psychoneuroses, tremors, sweats, cardiac arrhythmia, pupillary abnormalities, etc. One of the most difficult sequelæ to treat is that because of which the patient resembles a case of Parkinson's disease. He has the same mask-like facies, the same spasticity of the voluntary muscles, the voice, and the gait of a case of paralysis agitans. He may have a marked tremor also, but it is not that typical of Parkinson's disease, since as a rule it is increased on voluntary movement. Other patients are left with choreiform, athetoid, or myoclonic movements. Still others have definite psychoses, while still others have various paralyses, neuralgias, deafness and vertigo.

The mortality of this disease is at least 20 per cent. Many recover in from six months to two years, but about ten per cent of these are left with a progressive disease of the central nervous system.

So far as we know the only **early treatment** of epidemic

encephalitis which has value is drainage of the spinal fluid by frequent lumbar punctures, and hyoscine and adrenalin administered hypodermatically. The patient with this disease should rest quietly in bed, protected from all disturbing sensations, should be well fed, and carefully nursed. Physical therapy for cases with paralyses may have value.

Tetanus

Tetanus or "lockjaw," an all too common malady, is due to a specific germ, Bacillus tetani (Fig. 128), which enters the body through wounds made by dirty instruments, and especially those received in a stable. This bacillus is a normal inhabitant of the intestines of cattle, and hence is always present in fertilized soil. In some localities tetanus is the cause of death of over half the newborn children, because of improper cleanliness when caring for the navel. This is the germ which has made Fourth of July celebrations such serious matters. The symptoms of tetanus are due to the toxin of the tetanus bacillus. The bacillus itself stays in the wound. It does not, like most germs, invade the blood and organs, and its toxin travels, not in the blood stream, but along the nerves.

The period of incubation of tetanus varies from five days to five weeks after the injury. Then begin stiffness of the neck and difficulty in moving the lower jaw. **This muscular stiffness** increases and extends over all the muscles of the body. The face has the sardonic smile, due to the unequal strength of the facial muscles all of them in spasm. In addition to their stiffness the muscles are subject to convulsive spasms during which the whole body is so rigid that the patient is unable to move a muscle, to speak, or even to breathe. He may, therefore, suffocate. Because the extensor muscles of the back are stronger than the flexors, the spine, during a convulsion, is held stiffly in so convex a position that the patient rests on his head and heels. These spasms may follow an even slight stimuli, like a noise or a touch. It is important in diagnosis that between these paroxysms the body relaxes but very little.

Over 80 per cent. of these patients, if untreated, die; by far the majority of them, within four days from the onset of symptoms. The earlier these appear after the wound the more serious is the prognosis.

To prevent tetanus the wound should be **opened at once** and thoroughly cleaned out, and the patient should receive **a prophylactic dose** of anti-tetanus serum (500 to 1500 units).

After the symptoms of tetanus have begun the patient is kept in a dark room, and as **quiet as possible.** To quiet him one uses phenobarbital (luminal), 3 grams each four hours by mouth or rectum, or morphia, or even chloroform. Some inject magnesium sulphate solution subcutaneously (2 c.c. of 25 per cent. solution per each 20 pounds of body weight each six hours) or intraspinously (0.5 to 1 c.c. of a 25 per cent. solution per each 20 pounds of body weight).

The tetanus antitoxin is very effective in preventing the disease, but is less so in curing it after the symptoms have begun. Yet, large doses, over 100,000 units in all, are injected during the first three days after the onset. First, to desensitize the patient, one injects subcutaneously 1 c.c. of the antitoxin. If no reaction follows (dyspnea, cyanosis, or rapid pulse) 20,000 units are injected intravenously thirty minutes later, and at the same time another 20,000 units intraspinously. If, on the other hand, a reaction did follow, one delays the next injection three hours. These injections are repeated on each of the following two days.

Gonorrhœal Infections

Gonorrhœal infections, caused by the gonococcus (Fig. 127), are characterized by their latency and chronicity. For years they disturb the health very little, seldom causing death, but in the long run they are among the most important and destructive of diseases. **Gonorrhœal urethritis,** the local primary trouble, seems trifling, merely a discharge which lasts but a few days, but the infection may persist almost without symptoms for many years, leading in men to stricture of the urethra and prostatitis and in women to **pelvic inflammatory disease** and pelvic peritonitis. For both sexes this is the com-

monest single cause of sterility. The gonococcus explains almost half of the cases admitted to the female surgical wards. One of the commonest forms of **chronic arthritis** is due to the gonococcus. It may invade the blood causing a fatal septicæmia and one of the worst forms of valvular heart disease. Formerly it explained more than half the cases of **blindness of infants,** but this is now prevented by the drops of silver nitrate put into the eyes of every newborn baby.

Acute Rheumatic Fever

Acute rheumatic fever (acute articular rheumatism) is an acute, non-contagious fever caused by some germ, or by its toxin, (but what that germ is no one knows), and which is characterized by acute transitory polyarthritis, or acute chorea, or acute simple endocarditis, or erythemia nodosum. The disease tends to recur, and in any one of the attacks may manifest itself as one, two, or more of these conditions.

Acute articular rheumatism, the name often used when the attack affects the joints especially, is an acute and intensely painful arthritis which may affect several joints in succession, but which, on subsiding, leaves them normal. It is a disease of temperate climates, and over half of the cases develop during the first four months of the year. It attacks persons of all ages, but especially those between fifteen and twenty-five years of age.

The attack, as a rule, **begins abruptly.** The temperature rises rapidly as the joints become swollen, and within one day the disease is well developed. It is usually a polyarthritis, that is, more than one joint is affected. These joints become **swollen, red, hot, and exquisitely painful.** The patient cannot move them in the least, the slightest weight of the bedclothes is unbearable, and the patient, watching the nurse and doctor as they approach, begs them not even to touch the bed. **Children,** however, **suffer much less.** It may indeed be hard, even when their joints are acutely inflamed, to keep them in bed.

The joints most often involved are the knee, ankle, shoulder, wrist, elbow, hip, hand and foot. Only a few joints are

inflamed at one time, but the disease travels from joint to joint, one swelling as the last subsides. Each joint recovers completely. Not even does a slight stiffness remain permanent. **The prostration** caused by this disease is extreme. **Profuse acid sweats** with a sour odor are a disagreeable feature of some cases. The convalescence is slow and the disease is very **apt to recur.**

Simple endocarditis (page 105) and **Sydenham's chorea** (page 352) may be considered as complications and sequelæ of acute rheumatic fever or as different manifestations of the same disease.

Acute articular rheumatism seldom kills a man (the mortality is less than 3 per cent.) but **simple endocarditis** fills a large number of our hospital beds. To use Dr. Osler's expression, "rheumatism (i. e. acute rheumatic fever) kills at long range." The first attack of acute rheumatism leaves over half the cases with heart disease; the second, over 60 per cent; and the third, over 70 per cent. **Pericarditis** follows rheumatism oftener than it does any other acute disease. **Tonsillitis** also was formerly considered a complication of this disease, but now it is recognized more as a primary factor, and the present conviction of clinicians is that, were the tonsils properly treated, the number of cases of rheumatism, heart disease, and chorea, would be greatly diminished.

The treatment of acute articular rheumatism is to make the patient comfortable and to prevent complications. Acute rheumatism is a self limiting disease, and probably no drug will shorten its course. The tonsils, even if but slightly enlarged, should at once be removed. The bed should be as comfortable as possible. The patient should lie between blankets to avoid chilling, and should, on account of the sweats, wear a flannel garment.

The diet should consist chiefly of milk diluted with alkaline waters. Meat is excluded. Large amounts of water should be drunk. The sore joints, gently rubbed with oil of wintergreen, may be well wrapped in cotton and with the aid of pillows kept in comfortable positions or immobilized

by splints. Their pain may be relieved also by very hot or very cold compresses, or the Paquelin cautery. **Salicylic acid** is the best drug to relieve the pain. To be successful this may be given in large doses, almost till its toxic symptoms begin. The best form is sodium salicylate (in 15 to 20 grain doses every hour, or 120 to 180 c.c. of a 2 per cent solution three times a day by rectum) or acetyl-salicylate (aspirin) in 1 to 1.3 gm. doses. After the toxic limit (deafness, tinnitus, nausea, vomiting, etc.) has been determined one continues the drug, but gives daily a slightly smaller dose, until all signs of infection have been absent for ten days. Smaller doses of salicylic acid would seem to give little benefit; too large doses sometimes cause mental symptoms.

The nursing of the convalescence of acute articular rheumatism is most important. Many cases of valvular heart disease doubtless could have been prevented had the patient not got up too soon. Even when no complication is evident the patient should stay in bed six weeks after he feels well, a difficult matter in the case of children, yet this is important since they have a greater tendency to heart involvement than has the adult. The patient should then get up slowly, first into a chair, then walking, but always controlling the amount of exercise by its influence on the pulse rate. Any increase in pulse rate which persists after exertion for more than five minutes is accepted as indicating that the exercise was excessive. If any sign of heart involvement appears the patient should stay quiet much longer, an ice bag over the heart (page 107).

Many cases of true acute rheumatic fever, especially in children, run a course which is so very mild, that is, so **subacute,** that the patients object to staying in bed. Indeed, the inflammation of their joints may be very slight, but these patients have about the same danger of heart complications as have those with severer cases, and the treatment of both should be the same.

Almost any illness characterized by pain and swelling in or around the joints is spoken of as "rheumatism," but the

term "acute rheumatic fever" is now limited to cases which answer the above description; all other cases are spoken of as "acute arthritis."

Arthritis

Secondary arthritis, or infectious arthritis, is the name applied to that large group of cases in which the organism which causes the trouble can be isolated from in or around the joints. Arthritis can be a complication of almost any infectious disease, but the germs most frequently responsible are Bacillus tuberculosis, streptococci, staphylococci, the gonococcus, and the meningococcus. The first attack of an acute secondary arthritis (e.g., that due to gonorrhea) may resemble acute rheumatism in its onset and course, also in the fact that it may leave many of the inflamed joints clear; but, as a rule, at least one of them remains more or less permanently injured. The pyogenic germs may cause destructive abscesses in the joints.

The treatment of acute infectious arthritis depends on the germ. A little of the joint fluid should be removed with a syringe and cultures made to determine the organism. If the fluid is cloudy, that is, purulent, the joint should be opened and the fluid emptied. A tuberculous joint, however, is kept perfectly quiet and the patient given the full treatment for pulmonary consumption. Other cases improve under the hot-air treatment. If the joint shows signs of stiffening, then massage, passive movements, and active exercises may check this; but if these fail, one must at least prevent ankylosis in an unfortunate position. A stiff knee should be fully extended; a rigid elbow or ankle, on the contrary, should be bent to a right angle.

Chronic rheumatism is the very uninforming name given to all conditions marked by chronic stiffness, or pain, or swelling of the joints. The patients usually are elderly persons and those whose work exposes them to cold and damp; hence it is very common among the poor. The pain in such cases is worse during cold, damp weather. The question whether

or not these conditions are related to acute rheumatic fever is still unanswered.

The best treatment of chronic arthritis is massage, forced motion, and the Paquelin cautery; among drugs, aspirin and potassium iodide are useful. Changes of climate and a stay at the various watering places, if possible, are very beneficial.

The terrible disease, **arthritis deformans,** also called "rheumatoid arthritis" (although it may not be at all related

FIG. 154.—Atrophic arthritis of hand, causing marked trophic changes.

to "rheumatism"), is, many think, caused by germs, but which germs is not known. In some cases the small joints especially are affected, in others the larger joints only, in some the spine only, and in still others the spine and large joints. In all cases, however, some at least of the joints suffer permanent and sometimes serious injury. Two general groups are recognized: those with considerable hypertrophy of the tissues in and around the joints (the **hypertrophic** form), and, second, those with thinning of the bones and cartilages around the joints (the **atrophic** form). Usually both processes are present, but in different degrees. In the former there is considerable increase in both the soft and hard tissues of the

joints; the synovial membrane becomes very thick, the capsule becomes dense, and spicules of bone may surround the joint, with, as a result, limitation of motion or a complete locking called **ankylosis.** Sometimes the whole spine becomes one rigid bone, a condition called "poker back"; sometimes it is the hips, the shoulders, or the fingers that become immovable; some unfortunate persons can scarcely move a single one of their joints. This is a disease of adult life especially. Sometimes these forms of arthritis begin insidiously, but more often with an acute attack which differs little from acute rheumatic fever. In either case the patients lose weight and strength, the joints gradually stiffen, and crackle (crepitation) when moved. The joints gradually increase in size, but may appear to swell more than they really do because the muscles around them have atrophied. The pains are sometimes very severe; sometimes there is none at all. As the joint stiffens it may assume some unfortunate attitudes called **contractures.** Later, the acute disease ceases, and then the patient, except for the stiffness of the joints, enjoys fair health.

Those cases which **from the first present atrophic changes** may manifest few signs of acute infection. These, and especially the ones which begin near the menopause, seem due in part to disturbances of the internal secretions. Others, however, suggest such a lack of resistance to infection on the part of the joints that atrophy only follows their disease.

In the **treatment of arthritis deformans** all foci of infection should be removed or drained; especially diseased tonsils, infected teeth, and infected nasal sinuses. The patients should be given a full diet, certainly none of the anti-rheumatism or anti-gout diets or medicines. They need fresh air and sunlight. Hydrotherapy, especially warm baths, or a stay at Hot Springs, may help. Massage, passive movements, active exercises, and any means to increase mobility and to prevent ankylosis in unfortunate positions, are important. Baking, diathermia, and all physical therapies which increase the circulation to the joints, which reduce the involuntary spasm of the muscles, or which increase the range of motility of

the joint should be used. Their greatest value, however, would seem to be that immediately after their administration the joints allow more vigorous active and passive exercises than before.

Muscular Rheumatism

The term **muscular rheumatism,** which means little to the doctor, is conveniently applied to any indefinite painful condition of the limbs, not located definitely in the joints. Certain forms are common, as lumbago, stiff neck, and pleurodynia (pain felt in the muscles of the chest on each respiration), but there are many other muscular aches and pains which differ greatly in severity. Many of these we know are due to those infections of the spine which involve also the spinal nerves, producing pains which radiate to the terminals of these nerves in the trunk. In spondylitis of the lower thoracic and lumbar spines the pains in the anterior abdominal wall are often mistaken for appendicitis, gall-bladder trouble, etc. Others are referred from the diaphragm to the shoulder. The cause of others we do not know, except that they follow a wetting or exposure to cold—a draught, for instance—or hard muscular exertion. An accurate diagnosis is the first requisite for treatment. For immediate treatment, rest is the important thing, aided by hot compresses or dry heat, physical therapies, and aspirin.

CHAPTER XXI

Other Diseases Caused by Bacteria

Tonsillitis

The tonsils are two almond-shaped bodies located one on either side of the pharynx. In addition to these are the adenoid (Fig. 61) at the roof of the pharynx, the lingual tonsil at the base of the tongue, and many tiny ones, all of which form a ring around the throat. During childhood the tonsils are large, but they diminish in size with age. While we do not know their exact function, they seem to be protective organs.

Acute follicular tonsillitis is an inflammation of the tonsils caused usually by streptococci. These organs, usually both, are much swollen. On the surface of normal tonsils can be seen the mouths of the crypts. When acute tonsillitis is present these crypts contain little abscesses, whence the name "follicular" tonsillitis, which can be seen as little white spots from which a little stream of pus often trickles down.

An attack of tonsillitis is usually the result of exposure to wet or cold. The fever for a few days is high, and accompanied by headache, much malaise, pain in the joints, pain on swallowing, sore throat, and swelling and soreness of the lymph nodes at the angle of the jaw. There are few diseases which make one feel so wretched. The attack lasts about a week.

The other tonsils, including the adenoid, may suffer in a similar way. The lingual tonsil when inflamed gives intense pain when the patient swallows.

Tonsillitis is a disease of young persons especially, but also of adults. It occurs in epidemics, especially in crowded institutions, and most often in the early spring and fall.

As **treatment,** a good dose of calomel should be given at once. An ice-bag or cold compresses on the throat will give great relief. The best authorities condemn the use of gargles, swabs, and other local treatments. The patients find great relief in sucking ice and should drink large quantities of cold

water. Salicylic acid, aconite, guaiacum, and Dover's powders are useful.

In follicular tonsillitis the abscesses in the crypts are superficial and discharge into the mouth, but in **suppurative tonsillitis, or quinsy,** the pus is so deep in the tonsil that it cannot discharge externally, and so collects as one large abscess underneath the tonsil. This may push the tonsil and pharyngeal wall even past the midline of the throat. When both tonsils are similarly affected they may actually close the throat. The temperature is high and the prostration is

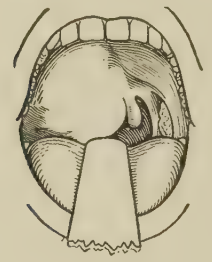


Fig. 155.—Throat showing quinsy on the right side and resulting distortion of the soft parts.

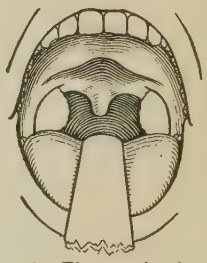


Fig. 156.—Throat showing chronically enlarged tonsils.

extreme. So far as symptoms go there is scarcely a more distressing disease. If let alone the abscess will in time rupture, but it is better to lance it early.

Chronic tonsillitis, that is, chronically infected tonsils, often without sore throat, may follow an acute infectious fever, such as scarlet fever, diphtheria, or an acute tonsillitis. In the most of these cases the tonsils are buried, that is, as a result of disease or of local treatment (cautery) the orifices of its crypts have become sealed and the pharyngeal pillars become adherent to their surface, with the result that no pus can escape and so cause sore throat. The infection in the closed crypts, therefore, discharges itself into the blood stream even for years after the patient has had his last sore throat. These are the tonsils which keep alive many cases of pulmonary tuberculosis, endocarditis, nephritis, arthritis, etc.

The importance of removing chronically infected tonsils, no matter how small or deep, is now fully realized. They should be dissected out, not clipped off.

The effect of adenoids on the mechanics of respiration is discussed on page 142.

Pyorrhœa

During the last few years the infection of the gums around the teeth, **pyorrhœa alveolaris,** and particularly the infection of the alveolar processes around the roots of dead teeth (root abscesses) have been considered responsible for the production of chronic diseases of the kidneys, blood-vessels, heart, joints, etc. Some feel that all dead teeth should be pulled since sooner or later it is likely to become the source of infection. All agree now that it is necessary to clean up carefully all pyorrhœa and not only to remove a tooth around whose root the X-ray films show that there is disease, but also to curette out the dead bone from around its socket.

Vincent's Angina

Vincent's angina, ulceromembranous stomatitis, or trench mouth, is seen especially among the soldiers living under debilitating conditions, among the poor whose food is bad and whose personal hygiene is poor, but also among all with debilitating conditions, and those with carious teeth. It is due to the combined infection of Bacillus fusiformis and Vincent's spirochæte. It may be limited to the gums or involve also the inner surface of the mouth, tongue and the tonsils. The disease begins at the margins of the gums, especially around the incisors of the lower jaw, from thence it spreads to the gums of other teeth. The gums first become swollen and ooze blood, and later their inflamed borders become necrotic and present against the teeth an ulcerated margin covered with a yellowish slough which may spread even to the alveolar processes, causing necrosis of the bone. There is a putrid odor to the breath. There is a marked salivation. The neighboring lymph nodes are enlarged and tender.

The diagnosis of the disease is made from smears from the exudate from the infected gums. Carious teeth should be pulled and infected tonsils removed. The mild cases may recover after the use of tincture of iodine, or of 10 per cent silver nitrate applied locally, or of a potassium manganate mouth wash, 1 to 200. The severer cases respond to arsenic given as Bowman's solution or as arsphenamine applied locally, or to oxygen-producing applications directly on the gums. Arsphenamine also may be given intravenously.

Agranulocytic Angina

Agranulocytic angina is a very serious tonsillitis and gangrenous stomatitis which begins acutely with fever, necrotic ulcers in the mouth, slight jaundice, extensive leucopenia without granulocytes and no skin hæmorrhages. The average leucocyte count is 1200 cells, the lowest, 100 cells per cmm., with small mononuclears even 100 per cent of the total count. The red cells, hæmoglobin and platelets are normal. This disease occurs especially in women (78 per cent) in middle life. Many different germs have been recognized as the cause, but whichever it is, it is one which attacks the bone marrow.

Glandular Fever

Glandular fever or infectious mononucleosis is a disease of children especially, which develops suddenly, with pain on moving the head and neck, sometimes nausea and vomiting, redness of the throat (the tonsils may be a little red but the throat symptoms are transient and unimportant), high fever, swelling and tenderness of the posterior cervical lymph nodes, and a leucocyte count of even 20,000, due to an increase of the mononuclear cells. The lymph nodes most often involved are the posterior cervical, the lingual, and the axillary. Their swelling is evident on the second or third day. They may vary in size from a pea to a goose egg, and are very painful. Pain in the chest and paroxysmal cough suggest involvement of the tracheal and bronchial nodes also. The liver and spleen as a rule are enlarged and tender. The treatment is symptomatic.

Botulism

Botulism. A few years ago we heard much about "ptomaine" poisoning. By "ptomaine" was meant a poison which is furnished by the decomposition of foods by bacteria. Food in this condition, however, does not develop such poisons, and yet food poisoning is a real and serious matter; but the poisons are the toxins of those germs themselves which are spoiling the food. The best illustration of this is botulism. A very serious disease due to the toxin which Clostridium botulinus liberates when it grows in foods in the absence of oxygen. It develops chiefly in home-preserved foods and meats, seldom in those prepared by commercial houses, for the latter sterilize their products at a temperature which kills all spores. The germs therefore multiply and later the canned food is so poisonous that persons have died who have merely tasted it. This poison, much like curare, attacks the nerve endings in the muscle fibers. The patient has no pain and is mentally clear, but if the case is severe he gets weaker and weaker, soon he cannot swallow, cannot cough, cannot talk and later cannot breathe. In America about 65 per cent die. Among the cases which recover, however, are some mild ones. The symptoms begin hours or days after the food is eaten. The fatal cases die before the fifth day. An antitoxin has been prepared which is successful if given early. To prevent this disease all doubtful preserved food should be heated immediately before eaten, since the boiling temperature will destroy the toxin. If, however, the food thus heated is allowed to stand, more toxin will rapidly form and in twenty-four hours the food will be as poisonous as before.

Tularemia

Tularemia, a disease of wild rodents, especially rabbits, and caused by Bacillus tularense, is very easily contracted by those who in any way handle infected animals. The disease is very contagious. An ulcer forms at the point where the poison enters the body, the neighboring lymph glands become enlarged, and then follows a long irregular fever. All get well. The diagnosis is best made by the agglutination test.

CHAPTER XXII

Unusual and Tropical Diseases Due to Bacteria

Leprosy is a chronic infectious disease caused by Bacillus lepræ, an acid-alcohol-fast germ which in many ways resembles Bacillus tuberculosis. As in tuberculosis, the characteristic lesions of this disease are small tumors (tubercles) which in leprosy develop especially in the skin and along the nerves. **The earliest lesions** would seem to **appear in the nose,** and the earliest diagnosis is often made by the discovery of these germs in the nasal secretion.

Leprosy is very common in Asia, is not infrequent in Scandinavia, Iceland, and the Sandwich Islands, while there are about 500 cases in America. It is not a very contagious disease. The fear of it in Eastern countries is explained by the fact that Orientals call many different skin diseases "leprosy."

There are two varieties of leprosy. **The tubercular variety** is characterized by the appearance in the skin of lesions which first are pigmented (later white) spots, and which later develop into little nodules, which slowly ulcerate, and then heal, leaving deforming scars. The hair, eyebrows, and eyelashes fall out. The sight is soon lost. The fingers and toes may ulcerate and drop off. These numerous lumps, open sores, and puckered scars give the patient a hideous appearance.

In the anæsthetic variety tumors similar to those described above grow in the nerves, as a result of which the tissues which these nerves supply become numb and atrophy. Patients with this variety of leprosy for years may not show any conspicuous signs of the disease.

Segregation is necessary since the sputum and the secretions of the nose and ulcers spread the germs. Countries in which leprosy was common have in this way practically stamped the disease out

Chaulmoogra oil, given by mouth in doses of 5 to 60 drops of the pure oil three times a day; or, better, given subcutaneously (from three to four injections each week of from 5 to 60 drops of a mixture containing chaulmoogra oil 60 c.c., camphorated oil 60 c.c., and resorcin 4 gms.) two or three times a week, is proving a valuable remedy for this disease. Some cases would indeed seem quite cured.

Glanders is a disease of horses especially, which man may contract by accidental infection. It is caused by a specific

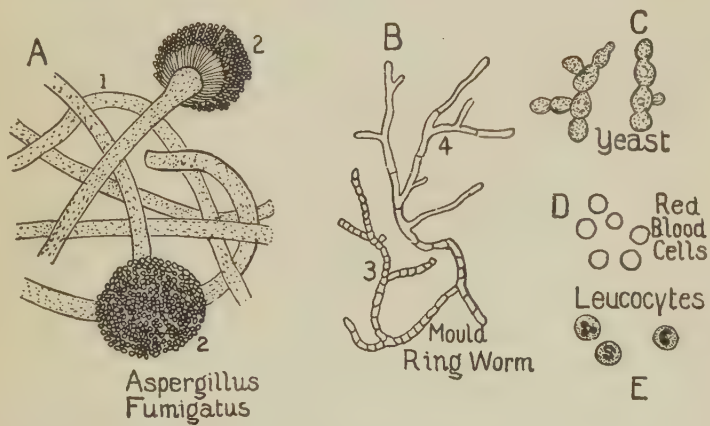


FIG. 157.—Some other important vegetable parasites. *A*, a mould; 1, the mycelial threads, 2, the fruit head. *B*, the parasite causing ring worm; 3, threads which are dividing into spores from each of which a new "plant" can grow; 4, threads not sporulating. *C*, a common yeast. *D*, red blood corpuscles, and *E*, leucocytes, introduced for comparison of size. All are drawn to the same scale. Magnified 250 times.

germ, Bacillus mallei. The infection begins in the nose as a multitude of little tumors which rapidly ulcerate, giving rise to considerable offensive nasal discharge. When these nodules lie under the skin, the more common form in man, the disease is called **farcy.** In the acute form of farcy these nodules, the farcy buds, soon suppurate, leaving ulcers. Abscesses also form in the muscles and in the joints.

Acute glanders may be fatal in eight or ten days, but chronic glanders may last for months, its only symptom a chronic coryza. Such cases, however, are very apt suddenly

to become acute. The acute form of farcy is practically always fatal; very chronic cases may get well. The only treatment possible is to excise all of the lesions.

Asiatic cholera is a disease of the lower ileum especially, caused by a specific germ, Vibrio choleræ (Fig. 129). It is endemic in India and has been epidemic in other countries, including our own. Its most important symptoms are severe diarrhœa and collapse.

The germ enters the body through the mouth and multiplies enormously in the intestine. The organism itself does not invade the body, but its toxin, which is absorbed through the intestinal wall, causes the disease. That cholera is not highly contagious is indicated by the fact that few nurses and doctors who have attended cholera patients have contracted it.

Cholera is spread by drinking water polluted by the stools of persons afflicted with this disease. Perhaps flies also aid, since they feed on these stools and then infect our food. Those handling the soiled linen of cholera patients often contract the disease.

The incubation period of cholera may last from a few hours to five or six days. The attack usually begins with **diarrhœa,** colic, and considerable malaise. These symptoms last a day or two, and then comes the **stage of collapse,** which lasts from two to twenty-four hours, with profuse diarrhœa and incessant vomiting. The blood would seem to pour all of its water into the intestine to wash out the toxins, therefore the thirst is excessive, the body visibly shrinks, the cheeks become hollow, the eyeballs sunken, the skin over the body becomes cyanotic and wrinkled, the secretion of urine stops, and the pulse becomes very weak. The intestines are soon washed clear of any fæcal matter and bile, after which time the profuse stools and vomitus consist of the water from the blood, in which is suspended many little white particles consisting of mucus and intestinal ephithelium, which give these movements the name **rice water stools.** Although the temperature is high the skin feels cold and clammy. Its general

color is ashy, and the hands and feet are blue. The blood pressure is low, and the blood count is high because of the concentration of the blood. The cramps of the muscles are terrible.

If the patient survives the stage of collapse the **stage of reaction** follows, during which the diarrhœa diminishes, the warmth of the body returns, and the kidneys resume the secretion of urine.

Individuals vary greatly in their susceptibility to this disease. Probably only a few of those who during an epidemic are exposed to cholera really contract it, and among those who do, all degrees of severity occur. Some have for a day or so only a mild diarrhœa—nothing more; while at the other extreme are those who die before the diarrhœa even has time to begin. Practically every severe case which recovers shows all the signs of nephritis and later even of uræmia. The mortality of the various cholera epidemics has varied from 30 to 70 per cent.

Prophylaxis consists in the isolation of the patient and the disinfection of his stools, vomitus, and bed linen. **The treatment** consists in keeping up his strength by stimulants administered hypodermically, especially by the massive intravenous injections of physiological salt solutions (2 liters each eight hours for one or two days) to restore the water of the tissues, and by the administration of large doses of sodium bicarbonate (even 90 gms. in 24 hours) to restore the salts lost to the body. Warm baths, warm blankets, hot water bottles, and large enemata to wash out the bowels are of value.

Bubonic plague, or the plague, is caused by a specific germ, Bacillus pestis (Fig. 128). The characteristic features of this disease are: the swollen inflamed lymph nodes, called buboes, the carbuncles, sometimes the fatal pneumonia, and, often, the hæmorrhages.

This terrible epidemic, **black death,** the most fatal of all acute diseases (it has a mortality of from 80 to 90 per cent) once swept over Europe, killing a quarter of the population.

Even now in Asia it is killing thousands of persons each year. An occasional case reaches our Northern seaports. The germs are spread by the fleas of rats, and the problem of preventing epidemics of this disease is the problem of destroying rats.

At the onset the patient suffers from "headache, backache, stiffness of the limbs, a feeling of anxiety, of restlessness, and great depression of spirits." The temperature rises for three or four days, drops slightly, then rises again to a higher point than before. Fatal cases usually die in extreme collapse during this secondary fever.

The buboes, or swollen inguinal glands, appear between the first and third days. In cases of pestis minor, they may disappear, but as a rule they suppurate and the abscesses break through the skin. This indeed is nature's surgery which may save the patient's life. Hæmorrhages under the skin are the rule. These are the so-called **plague spots,** or "tokens" of the disease, and when abundant gave the disease the name "black death." The spleen is enlarged and the leucocyte count rises even to 90,000 per cu. mm.

Some cases die from the septicæmia caused by the plague germs even before the buboes have had time to develop. The most fatal form of the disease is the pneumonia due to this germ (its mortality is 96.6 per cent). This also is the most communicable form for men.

The sputum, urine, and stools of patients may contain vast numbers of Bacillus pestis. The diagnosis is not difficult, because these germs are easy to recognize.

In treatment the most rigid isolation must be preserved. All evacuations must be destroyed, and all clothes disinfected. The patient should be made comfortable, and proper local treatment (ice compresses, surgical measures, etc.) should be applied to the buboes. Vaccination measures now in use promise to be of value. The dead should be cremated. The pneumonia form of the plague is so very contagious that all near the patient should wear masks, composed of gauze and raw cotton, entirely covering the nose and mouth. The other forms are not directly contagious.

Anthrax, the most wide spread and fatal of all animal plagues, and most prevalent among the sheep and cattle of Asia, Russia, and France, is caused by Bacillus anthracis (Fig. 128). This germ is spread by the grass the animals eat where the grazing land is infected by the bodies of animals killed by this disease and buried there.

Farmers, butchers, and those handling the wool, hides, or flesh of infected animals may contract anthrax. Where this bacillus enters the skin, usually a scratch, it causes a rapidly forming boil called a **malignant pustule,** which in a few hours after its appearance has developed a swollen periphery and a black gangrenous center. Cut out this boil at once, together with a good margin, and you may save the patient from fatal septicæmia. If the germ enters the body with the uncooked meat or milk of diseased animals it causes a rapidly fatal enteritis; if inhaled with dust it causes a fatal bronchitis, called wool sorter's disease, or rag picker's disease. So virulent are these diseases that the patients sometimes die in a few hours after the onset.

Undulant fever, formerly called **Malta fever** because its germ was first successfully studied on the island of Malta, is a noncontagious disease characterized by the undulatory course of its fever, arthritis, profuse sweats and enlargement of the spleen. It is caused by a specific germ, Micrococcus melitensis, which may be conveyed to man by the milk of infected goats, by the milk and flesh of the cow and by pork. It is so becoming so common that it is likely to replace typhoid fever in importance.

Its course, which lasts six months or more, consists of a **series of febrile attacks** with a definite inflammation of the joints, and profuse, bad-smelling sweats, each attack lasting from one to three weeks, and separated by a few days of apyrexia. The treatment is in general that of typhoid fever, but with special emphasis on a nourishing diet, an abundance of fluids to replace the water lost by the sweats, and, since the gums are spongy and bleed easily, mildly antiseptic mouth washes, frequently employed. The mortality of this disease is only about 2 per cent.

Typhus fever, called also hospital fever, camp fever, spotted fever, jail fever and ship fever, formerly "one of the great epidemics of the world," was, during the past great war, the worst of scourges for the half-starved peoples of Serbia, Russia and other inland nations. It is found wherever starving human beings are overcrowded amid filthy surroundings. It is a filth disease due, it is believed, to an organism named Rickettsia prowazeki which is spread by lice. For a long time no distinction was made between it and typhoid fever.

Briefly described, it is a very acute fever, with an incubation period of twelve days, a sudden onset with a chill, pain in the back and legs, and unusual prostration. On the third, fourth, or fifth day appears the macular rash, on the abdomen first, but in two or three days covering the entire body. Even in the mildest cases one finds among the macules many tiny hæmorrhages or petechiæ, which give the disease the name of **spotted fever.** The temperature is continuous and high, even 107° F. All cases show profound nervous symptoms (hence the name "typhus," meaning a cloud), prostration, delirium, and even mania, and, in fatal cases, coma. It terminates by crisis at the end of the second week, followed by rapid convalescence.

Its mortality varies from 12 to 20 per cent. The fever seems to have been especially fatal to the doctors and nurses attending these patients.

Doubtful cases may be diagnosed by an agglutination test similar in many ways to the Widal test for typhoid fever. The treatment is that of typhoid fever, with special attention to an abundant supply of water and to hydrotherapy.

Rocky mountain spotted fever, or tic fever, is an acute infectious disease met with in certain of the Western States, especially Montana and Idaho, which is conveyed by tics, and characterized by fever with chills, general pains, and a macular rash which becomes hæmorrhagic. The incubation period lasts from three to ten days. The onset is sudden, with a chill, high fever, and severe pains, especially in the arms and legs. On the second day, or soon after, there appears, first on the

extremities and later on the body, an eruption of macules which gradually becomes darker, and, finally, definitely hæmorrhagic. The patient may bleed also from the mucous membranes. A moderate leucocytosis is the rule. After about four weeks the fever terminates by lysis. The treatment is symptomatic.

Foot and mouth disease, epidemic stomatitis, or aphthous fever is a very contagious disease of animals with cloven hoof, due to an ultramicroscopic virus which is transmitted to man by drinking the milk of, or by contact with, infected animals. The onset of the disease is attended with malaise, fever, and an eruption of vesicles in the mouth and pharynx which rupture, leaving shallow ulcers. The course continues from ten days to three weeks. The mortality is about 8 per cent. The patient should be isolated. The treatment is symptomatic for the fever, a mouth wash of potassium permanganate, and the local application of silver nitrate to the ulcers.

CHAPTER XXIII

Diseases Due to Animal Parasites

General Discussion

Most of the diseases already studied are caused by unicellular bacteria. Other diseases are caused by equally primitive animals, the protozoa. It is easy to study many of the bacteria since we can grow them so easily in glass tubes, but the majority of protozoa can only with difficulty be pre-

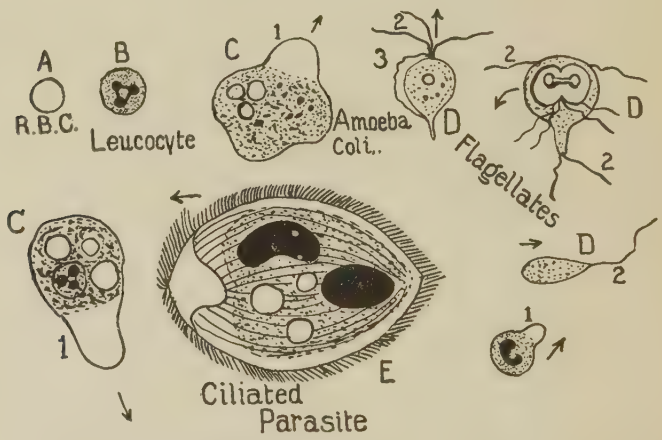


Fig. 158.—Some of the smaller animal parasites. *A*, red blood corpuscle, and *B*, leucocyte, introduced for comparison of size. *C*, entamœba histolytica, 1, pseudopod. The arrows in all cases show the direction of movement of the organisms. *D*, flagellated protozoa, 2, flagella, 3, membrane. *E*, ciliated protozoon. In the lower right-hand corner is a leucocyte in motion; note the contrast in size and appearance between this and *C*. In the lower left-hand corner is an amœba coli which has devoured red blood corpuscles and a leucocyte.

served alive in the laboratory. The others, we study best at the bedside. A few diseases, syphilis, amœbic dysentery, some forms of diarrhœa, malaria, the sleeping sickness of Africa, and yellow fever, we know are caused by these minute animals. This list, however, is increasing, and will doubtless increase still more, as our methods of work become more accurate.

Amœbic dysentery is caused by a minute protozoon. En-

tamœba histolytica, which resembles a leucocyte in many ways except that it is from two to ten times as big (Fig. 158, C). It is active, moving very rapidly under the microscope. Its nucleus, which is small and round, is seldom seen because the protoplasm of the cell is full, not of granules, as are the commoner leucocytes, but of fragments of food which the parasite has eaten—of leucocytes, red cells, bacteria, etc.

These amœbæ burrow into the mucous membrane of the colon and lower ileum. There they multiply. The result is a pus pocket with only a small orifice opening into the bowel, from which numerous burrows extend for considerable distances in all directions under the mucosa. Later the protecting roof of mucosa of this burrowing abscess sloughs off, or, tearing off at one end may hang as a rag, leaving exposed an ulcer, which from its shape and its method of formation is known as an **undermining ulcer.** The large bowel and the lower part of the small bowel may be so covered by these ulcers that very little normal mucosa is left. The depth of these ulcers varies. Usually their floor is the muscle wall of the bowel, but they may perforate the entire wall and cause a fatal peritonitis.

The chief symptoms of a case of acute amœbic dysentery are fever, often emaciation, and the passage of frequent small stools in which is much blood and mucus. If a speck of this mucus is examined it may be found to be swarming with these amœbæ.

The important features of amœbic dysentery are its chronicity, and the tendency of its course to consist of **recurring attacks** of acute dysentery separated by periods of constipation which last for a few months. Even during these periods of constipation the protozoa can be found in the stools. The amœbæ lie perhaps for years well protected in the bowel wall, quite out of reach of medicines or bowel irrigations.

The treatment of this disease is ipecac in any form, given in twenty-four day courses. Some give emetine hydrochloride hypodermatically, one grain twice a day for twelve days, but now the most popular method is to give emetine

bismuthous iodid (containing thirty per cent of emetine) three grains daily for twelve consecutive days. One should never give a patient more than eighteen grains of emetine in all, since toxic symptoms may appear (peripheral neuritis, diarrhœa, loss of weight, etc.). Others use arsphenamine intravenously, either alone, or, better, before or after one of the emetine courses.

The easiest way to diagnosticate this form of dysentery is find the living amœbæ and see them move. The protozoa on the slide soon cease their movements and are not easily to be recognized when quiet; everything, therefore, should be ready for the examination before the stool is passed. The nurse can be of great help to the doctor in this search. A dose of salts is given early in the morning, or on the evening before the examination, to loosen the stool and to wash down mucus from the upper colon. A vessel or bed-pan warmed to about blood heat (not a hot pan, for that would cook the parasites) may be used, or, better, a warm rectal tube is passed as far up into the rectum as possible. A little mucus will usually be found in the eye of the tube. One drop of a particle of mucus, especially bloody mucus, or of the liquid part of the stool, is picked up with forceps, spread on a warmed glass slide by the weight of the cover glass, and examined at once under the microscope.

The healing of the old amœbic ulcers may cause stricture of the bowel. The amœbæ in about one-fifth of the cases find their way into the portal veins and are carried to the liver (see page 246), where they cause abscesses. These abscesses differ from those due to bacteria in that they are more often single than multiple, are large, and have little evidence of real inflammation in their walls. They are merely large holes in the liver full of decayed and liquefied liver tissue. In their necrotic walls are hosts of amœbæ. These are the abscesses which may get well after perforating through the lung. One point to be emphasized is that not infrequently these abscesses are found unexpectedly in patients who have had few or no symptoms suggesting dysentery.

Amœbic dysentery is a disease of the tropics, especially Egypt, India, and the far East, but is common also in our Northern States.

Harmless amœbæ are common enough in stagnant water, and also may be found in the stools of normal persons (Amœba coli). The pathogenic amœbæ survive outside the body as encysted forms and enter a new host with his water or food. In the bowel these cysts again become amœboid forms and either at once attack the mucosa of the colon, setting up a colitis and resulting dysentery, or, not harming this host, are spread by him as carrier to other more susceptible persons.

Ciliata and flagellata, often found in the stools, seldom, if ever, do any harm, although when very numerous they do cause a diarrhœa which in some cases has proved fatal (Fig. 158).

Malaria is an acute infectious disease caused by protozoa which strongly resemble leucocytes, except that they are much smaller. Of these **malarial parasites** there are **three** slightly different **varieties,** all grouped under the general term Plasmodium malariæ. One of the three varieties is called the **tertian** parasite, because the cycle of its life takes just forty-eight hours and so ends on the third day. **The quartan** is so named because its cycle always takes seventy-two hours, and so is completed on the fourth day. **The æstivo-autumnal** parasite, so named because the form of malaria which it causes develops especially in summer and fall, has a cycle which varies between twenty-four and seventy-two hours. In the following pages we shall indicate, when necessary, the individual varieties by the terms tertian, quartan, and æstivo-autumnal, but when we use the term malarial parasite it will include all three (Fig. 159).

A malarial parasite **lives in a red blood corpuscle.** When young it attaches itself to a red cell and later makes its way to the cell's interior, where it is well protected from the hostile blood-plasma, as well as from medicines. There it grows, consuming the hæmoglobin as its food. When full grown

it fills the cell, which then is merely a hollow shell. The parasite next divides (segments) into from ten to twenty small parasites (called hyalines), the containing cell bursts, and these hyalines are scattered in the blood stream. For a short time, during which the most of them die, they are free in the plasma, but a few soon find their way into new red cells. There the process described above repeats itself.

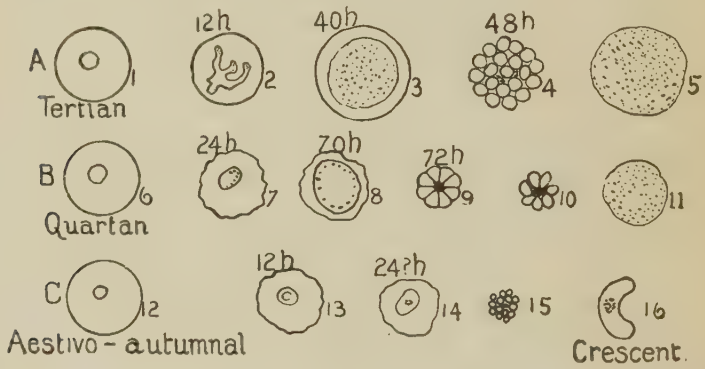


FIG. 159.—The malaria parasite. (Magnified 1000 times.) *A*, the parasite of tertian malaria. 1, a young hyaline which has just entered the red corpuscle. 2, the same 12 hours later. The parasite is larger, actively amœboid and now contains pigment granules. 3, the same when 40 hours old. The red corpuscle is swollen, the parasite is large and contains much pigment. 4, the same when 48 hours old. The red corpuscle has disappeared. The parasite has divided into twenty small parasites called "segments." These will separate and make their way into other red corpuscles, as was the case with 1, and start the cycle over again. 5, some of the parasites like 3 do not segment but grow to a form which will develop further only in the mosquitoes' stomach. There 5 will grow, divide into thousands of tiny forms, which if injected into a person's blood by the bite of this infected insect will like 1, start the cycle. *B*, the parasite of quartan malaria. Note the differences in ages, in size, the different effect on the red corpuscles which shrink, and the coarser granules of pigment. 9, the marguerite form, or "pre-segmenter" which divides to only about seven segments. 11, is similar to 5. *C*, the parasite of æstivo-autumnal malaria. Note the differences in ages, in size, in the effect on the red corpuscles, and the peculiar shape of the parasite, 16, which like 5, and 11, is designed for the mosquitoes' stomach. (The segments of 15 are drawn too small.)

The three varieties of malarial parasites differ somewhat in appearance, size, in the effect they have on the cell in which they live, and in the number of hyalines into which each adult divides, but we cannot dwell on these differences here.

The cycle of the **tertian parasite** takes forty-eight hours. In the blood of a patient with tertian malaria there may be hundreds of millions of these parasites in the body at one time, all exactly of the same age. They all divide (segment)

at just about the same time. It is just then that the **chill** and the **sharp rise in temperature occur,** and just then also that quinine, if present in the blood, will kill these parasites. A patient thus infected will, unless treated, have a chill every second day. He is said to have **single tertian malaria** since he has but one group of the parasites in his blood. Often, however, a patient has two groups of the tertian parasites in his

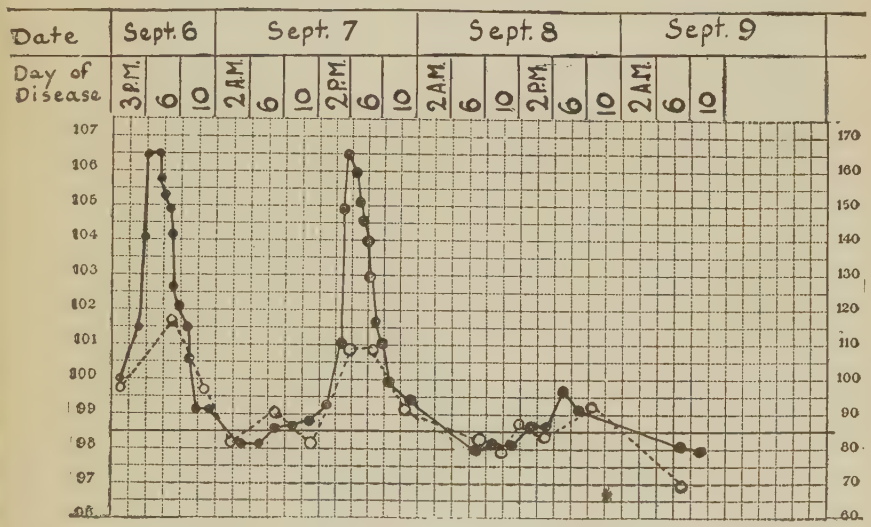


FIG. 160.—Temperature and pulse curve of a case of double tertian malaria.

blood at the same time, the one always twenty-four hours older than the other. This is like having two diseases at once, for the two groups are independent. Since each group will cause a chill each second day, the man with two groups will have **quotidian fever,** that is, a chill every day.

The cycle of the **quartan** is seventy-two hours long, and hence, if but one group is in the blood, the man will have a chill every fourth day—for instance, on Monday, Thursday, Sunday, Wednesday, etc.: if two groups are present in his blood the chills might come on Monday, Tuesday, Thursday, Friday, Sunday, Monday, etc.; if three groups, then he would

have a quotidian fever. The **æstivo-autumnal** parasites do not long remain all of the same age. At first, the patient may have a chill every day, but soon the parasites begin to vary in the time of their segmenting, and so the temperature will become more and more irregular, and the patient have only "dumb chills," or a continuously elevated temperature and no chills at all.

We have spoken thus far only of that form of the parasite's growth which causes fever in man. Some of the hya-

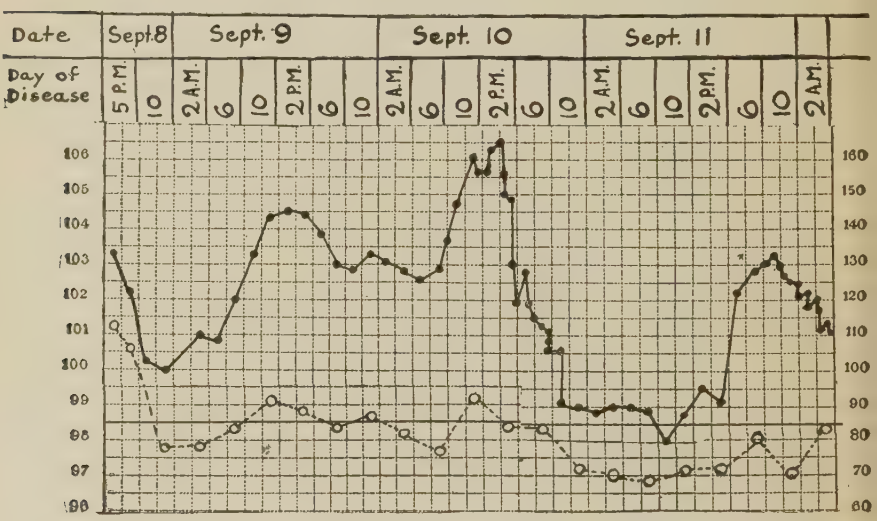


FIG. 161.—Temperature and pulse chart of a case of æstivo-autumnal malaria.

lines, however, mature but never segment. These remain unchanged perhaps for months, hidden in the spleen, liver and bone marrow. These are the **sexual forms.** They do the patient no harm, but if any of them happen to be in the drop of blood which a mosquito of the Anopheles group sucks out of that person they will mature in its body, producing, in a few days, myriads of young forms some of which that mosquito will inject into the blood of the next person it bites. For several days after that bite the new victim will have no symptoms, although during that time these parasites are increas-

ing rapidly in numbers, but some day they will be numerous enough to cause, first, chilly feelings, and later, real chills.

There is only **one variety of mosquito** which can act as the intermediate host between man and man, and that is the Anopheles. This can be recognized at a distance if seen when resting on a wall, for, instead of standing "hunch-backed," as does the ordinary mosquito (Culex), it stands with body, thorax, and bill in a straight line and forming with the wall an angle of about 30°.

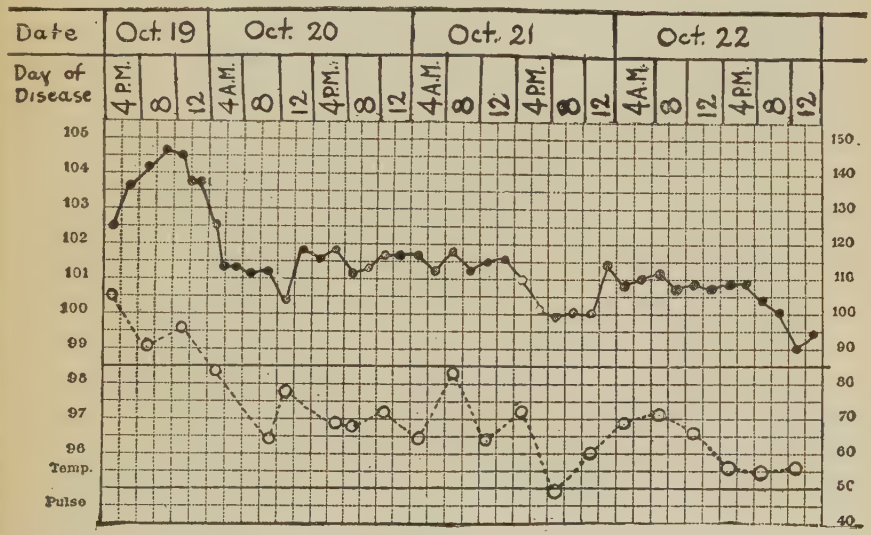


FIG. 162.—Temperature and pulse chart of a case of æstivo-autumnal malaria which suggested mild typhoid fever.

Patients with tertian and quartan malaria (the regular intermittent fevers) feel well on the days when they have no chill. **The chill** begins quite suddenly, with sensations of cold, shivering of the whole body, and sometimes nausea and vomiting. The face and hands are blue, and there is often intense headache. The temperature rises rapidly. After about ten minutes the patient warms up, and presently becomes intensely hot, his skin flushed, his heart beating violently, his head throbbing, and perhaps his mind delirious.

This stage lasts from thirty minutes to four hours, during which time the temperature reaches its highest point, from 104° to even 107° F. Then, with the prompt fall of the temperature to normal, the patient begins to perspire profusely, the headaches cease, he feels better, and soon is entirely relieved. The entire paroxysm lasts from ten to fourteen hours.

Æstivo-autumnal fever may at first cause a few chills, but the temperature becomes more and more continuous, and the chills, less and less severe than at first. Nevertheless, this form of malaria is much more serious than are the others, and causes far more pronounced anæmia. Very severe cases of any form of malaria may become comatose and die (**pernicious malaria**), or develop a severe nephritis, or a severe gastrointestinal disturbance, due to the accumulation of the parasites in the blood-vessels of the organs affected. Some patients pass large quantities of hæmoglobin in the urine (**black-water fever**).

To avoid malaria one must avoid the infected Anopheles mosquitoes. This insect bites usually only after sundown, never a moving object (hence the advice to those living in infected regions to sit in rocking chairs), and seldom through white cloth. Houses should be well screened, especially the room of a patient ill with malaria, in order that no Anopheles mosquitoes may become infected from him. Nowhere should be permitted little pools of stagnant water in which these mosquitoes can breed.

The diagnosis of malaria is simple; one must find the parasite in the patient's blood. Unless this is done, this diagnosis should not be made. Unfortunately, the term malaria is carelessly applied to any disease with chills. Cases of typhoid fever, of abscess formation, and especially of very early tuberculosis are often treated for malaria, which is unfortunate since valuable time is thus lost in beginning proper treatment. It may be said, in general, that any fever which a few consecutive doses of quinine does not stop, temporarily at least, is not malaria. On the other hand, the examination

of the patient's blood for the malarial parasite is important to avoid mistaking æstivo-autumnal malaria for certain conditions which, clinically, it may resemble more than it does the other forms of malaria.

The cure for malaria is quinine or arsphenamine. The patient should lie in bed as long as there is any fever, for malaria, especially the æstivo-autumnal form, is as serious as are any of the other acute infectious diseases, both as regards its own immediate dangers and those of its sequelæ. The quinine should be given in liquid form, because much of that given in pill form is not even dissolved in the intestine. In the severe cases the quinine is injected directly into a vein. The quinine should be given in regular doses, at first ten grains three times a day. The tertian and quartan fevers are best controlled by planning that the medicine shall reach the blood just before a chill is expected, since during a few hours the majority of the young parasites will be free in the plasma. The quinine should be continued, 10 grains three times a day for four days, then 10 grains each night before retiring for at least three months after all clinical traces of the disease is gone, for these parasites may for years hide in the bone-marrow and spleen, ready to cause a recurrence of the disease when the proper conditions arise.

Trypanosomiasis, called also "sleeping sickness" is a terrible disease of the tropics due to an animal parasite Trypanosoma gambiense (a small eel-shaped protozoön with long flagellum (Fig. 129, E) by means of which it swims actively among the red blood-corpuscles) which is introduced into the blood of man by the bite of a certain fly. Trypanosomiasis is a terrible scourge for the animals of India and Central Africa, in some parts of which countries cattle and horses cannot live.

After its inoculation by the bite of the fly this parasite multiplies in the blood, at first without symptoms, but later causing fever, loss of weight, and swelling of the lymph-nodes. Where, however, the parasite invades the central

nervous system it causes **drowsiness,** later coma, and, finally, death.

Arsenic (atoxyl), intramuscularly or subcutaneously, but better trypanosamial, seems to cure some cases if begun early enough.

Kala-azar, pyroplasmosis, tropical splenomegaly, dum-dum fever, a disease also due to minute animal parasites, the Leishman-Donavan bodies, is a tropical disease occurring especially in India and Egypt. How it is transmitted is not known. Its chief symptoms are an irregular fever, large spleen, anæmia, hæmorrhages, œdema, and leukopenia. About one-third of those infected die. These cases formerly were considered to be a special form of malaria which did not yield to quinine.

Syphilis (lues) is now considered of all diseases the worst enemy of the human race. It is caused by a parasite, usually considered to be a protozoon, but some say bacterium, Treponema pallidum (Fig. 129). This germ usually (but not always) produces at the point where it enters the body, and often four weeks later, a hard, painless, indolent sore which soon ulcerates, and which heals slowly, leaving a permanent scar. This is the **initial lesion,** or primary sore and is called a **hard chancre.** The chancre is sometimes so small that it is not noticed.

From six to twelve weeks after the appearance of the primary sore the **secondary stage,** or stage of general infection, begins. The patient then has a fever, becomes anæmic, complains of rheumatic pains in the bones, which are worse at night, has a chronic sore throat, his hair falls out, and often his eyes become inflamed. The most important feature of the secondary stage however, is a **skin rash,** which occurs in such a variety of forms that description would be useless. It is, however, most marked as a rule over the trunk, is painless, and seldom itches. It is, therefore, often overlooked by the patient. In the mouth and on the tongue also appear the **white mucous patches.** The patient, during this second stage, seldom feels ill, which is unfortunate since then the disease

is most easily communicated to others. Nurses should fear infection from these skin rashes and from the patient's saliva.

After the secondary stage, sometimes immediately, more often several years later, begins the **tertiary stage,** which is characterized by the formation of tumors called **gummata,** which are, in fact, large tubercles which soon break down. These gummata may develop in any organ of the body, but especially on the bones (most often on the shins, clavicles and skull), on the skin (therefore leg ulcers), in the cartilage of the nose, which as a result may collapse (resulting in a saddle nose), in the liver, in the wall of the bowel which may later develop a stricture, in the brain, where they give all the symptoms of tumor, but which disappear after courses of medicine, and in the wall of the aorta, one result of which is aneurism. Tertiary lues can simulate almost every disease of almost any organ, and if left without treatment may produce the worst of destructive effects. With proper treatment these tumors absorb rapidly, but may leave dangerous scars, weak spots in arteries, etc.

Years later locomotor ataxia or general paresis may end the sad picture.

As syphilis is supposed by many to be the result of immorality only, it is well to explain that many cases result from **the accidental infection** of the lips from kissing, or the use of common drinking cups, while nurses and doctors contract syphilis while examining or caring for cases, and hence have the primary sore on their hand or finger.

The first-born children of luetic parents usually are born dead or die within six months; and, of those which survive, the oldest often are mentally defective, or at least bear throughout life some physical marks of their inheritance.

The Wassermann test is of great value in the diagnosis of syphilis, since when positive the chances are that the patient has had lues. It, of course, does not prove that the present illness is lues. It is valuable also as **a control of the treatment** of syphilis, which should be continued actively until a positive Wassermann test becomes negative, no matter how well

the patient feels. Also, the reappearance of a positive test is good evidence that treatment should be resumed. When sterilizing the syringe which the doctor will use in obtaining blood for this test the nurse should be sure that it is chemically clean as well as sterile.

Arsphenamine (salvarsan, 606) was a great addition to the medicinal treatment of lues, nevertheless it is by no means a cure, and is now given in conjunction with even more mercury and iodides than were prescribed before its discovery. The best way to give **mercury** is the inunction of the metal (4 to 6 grams of the 50 per cent ointment is administered six times a week for six weeks) or the intramuscular injection of a soluble mercury salts (e.g. mercuric bichloride gr. 1/6, four to six times weekly). **The iodides** are given to the point of tolerance. (Sodium iodide, begin with 10 grains three times a day, and increase 5 grains a day till the patient is taking from 60 to 100 grains each day, greatly diluted in water). This treatment is continued for three years or until the Wassermann test becomes quite negative. The test is later repeated at intervals of six months each, and if it again becomes positive the treatment is resumed. Early in each case of lues the Wassermann test should be applied to the **spinal fluid** in order to determine early the need of intraspinous therapy. During the time that mercury is given, a **potassium chlorate mouth wash** should be used to reduce the danger of salivation, that is, of sore gums and an increased flow of saliva.

Dementia paralytica (general paresis) may be defined in popular terms as a very chronic luetic infection of the brain cortex which results in its atrophy. As a result, the convolutions of the cortex become thin and small and the meninges adherent to them. Both locomotor ataxia and general paresis are parenchymatous diseases, that is, are diseases of the nervous tissue itself, the former of that of the spinal cord, and dementia paralytica of that of the brain, while cerebrospinal lues is the term used of lues of the meninges and cerebral blood vessels. Dementia paralytica is a disease

of adult life. The commonest and most important early symptom is a **slight change in the personality** of the patient. He becomes careless, indifferent, unreliable. Some, but not many, become very egotistical and develop ideas of grandeur; others do unaccountable things; others commit without shame some foolish act; in still other cases a convulsion resembling epilepsy, or a "stroke of apoplexy" followed by only a temporary paralysis, are early signs. Later, one notices a tremor of the face, slowness of speech, and loss of memory. The patient becomes irresponsible, and, finally, completely demented. When the first symptom, usually a change of character, is noticed, a specialist should at once be consulted, and the spinal fluid examined. This gives early and reliable information. The cell count of the spinal fluid is usually a little increased (i.e. over six cells per cubic millimeter), its globulin is increased, often it gives a positive Wassermann test, and, especially, it gives a positive colloidal gold test, which is almost conclusive.

There are three methods of **treating general paresis**; the periodic injections into the spinal canal of the patient's own serum obtained from blood taken twenty minutes after an intravenous dose of arsphenamine; the intravenous injections of tryparsamide; and the production of an acute malaria. Just now the last is most encouraging. Into the patient's vein is injected blood from a person suffering from acute tertian malaria, and he is allowed to have a series of malarial chills. This is an illustration of how one acute disease controls another.

As soon as a paretic's mental features become pronounced he should be cared for in an asylum.

Tabes dorsalis (locomotor ataxia) results from syphilis of the spinal cord, its lesions limited chiefly to the posterior columns in which the fibers of joint sense run. Its chief symptoms are: loss of joint sense, "shooting" pains, loss of the reaction to light by the pupils of the eyes (the Argyll-Robertson pupil), and loss of knee kicks.

In tabes the muscles are not weak and there is no paraly-

sis, but the patient loses his joint sense, the sense by means of which we know without looking exactly the position of each limb. The patient is still strong, but he has a gradually decreasing power to control his movements. At first he walks along rapidly, with long, strong swinging steps, and a stamping gait, but only if he can see to watch each step; later he uses a cane to keep him from falling, then two crutches, and, finally, to stand erect he must be supported on both sides, and even then his legs get "all tangled up." Sometimes his arms are similarly affected. This is, this patient has ataxia, or "loss of order" of his limbs. The normal man walks almost unconsciously, his joint sense guiding the contractions of his muscles; but the man with tabes cannot trust these sensations and must use his eyes. This is the reason why he cannot walk in the dark, since then he cannot see to place his feet correctly. His skin sensations all are keen enough, and yet lying in bed he often "loses his legs"; that is, must feel for them to find out whether they are straight or bent. He stands erect with his feet together, but, if while doing so he closes his eyes, he sways or falls (**Romberg's sign**). He suffers sudden atrocious pains, especially in his legs, which are well described as **"lightning" or "shooting" pains.** He has also sudden attacks of pain in the abdomen with vomiting (**gastric crises**), and various similar severe bowel and bladder symptoms, which often lead to operations for gall-stones, appendicitis, etc. The **pupils** of the eyes **do not react to light,** and his knee kicks fail.

The treatment of locomotor ataxia is that of lues combined with rest and massage, and in addition the intraspinous treatment (mentioned on page 531). Some cases have improved wonderfully.

Relapsing fever is caused by a minute, spirally coiled, thread-like organism, Spironema recurrentis (Spirochæte Obermeiere) (Fig. 129), which, during the febrile periods, can be seen in the blood, moving actively among the red blood corpuscles. This disease occurs in India especially, but cases have been found in this country. It occurs espe-

cially among the very poor, who live in overcrowded hovels and are almost starving, hence the name "famine fever." It is actually, however, transmitted by infected lice or tics. When one of these is crushed on the skin the organism finds its way into the body through some scratch.

Relapsing fever occurs as **a series** (hence the name "relapsing") **of two, three or four sharp febrile attacks,** each lasting six or seven days, and separated by intermissions of about the same length. Each attack begins with nausea, vomiting, and pain in the back. The fever of these attacks rises suddenly, often with a chill. The temperature remains continuous and **very high,** and at the end of each attack falls by crisis. The disease is rarely fatal. This infection is practically cured by one injection of arsphenamine.

Yellow fever is an acute fever of the West Indies and Central and South America, the most characteristic features of which are: a very early jaundice (hence the name), an early albuminuria (due to acute parenchymatous nephritis), a pulse which early becomes slower while the temperature is rising higher, and a tendency to hæmorrhages, especially from the stomach, causing the well known "black vomit." The toxæmia it produces varies greatly in different cases.

There is good reason to believe that it is endemic and mild among the young negroes of the Tropics of the Western Hemisphere. When, however, it breaks out in a non-immune population it works terrible havoc. As an epidemic it has repeatedly swept over our Southern States and to as far north as New England. These epidemics develop in the warm, wet months, limit themselves to the seacoast, and are especially severe in the dirty, crowded districts of large cities.

The germ which causes yellow fever resembles somewhat that of syphilis, and is named Leptospira icteroides. It is spread by one **mosquito** only, Stegomyia fasciatus (Aedes ægypti) which may be recognized by his striped yellow-and-black legs. After one of these mosquitoes bites a patient ill with yellow fever at least twelve days must pass before that

mosquito cannot transmit the disease, but after that time its bite is dangerous.

The period of **incubation** lasts usually for three or four days after the mosquito's bite. The onset is sudden, beginning as a rule in the early morning hours, with chilly feelings, headache, and pain in the back and limbs. **From the very onset the face is flushed and slightly jaundiced.** The temperature rises rapidly, remains elevated from one to three days, then falls by a lysis which lasts two or three days (the stage of calm), and then rises again for the secondary fever, which lasts from one to three days. In fatal cases, however, the temperature remains elevated till death. Very early in the attack, while the temperature is rising, **the pulse becomes slower,** and at the height of the fever may have a rate of from 70 to 80, while during defervescence it may be as low as from 30 to 40 to the minute.

Albuminuria appears, even in the mildest cases, as early as the third day. In severe cases the nephritis may dominate the clinical picture.

Vomiting is the rule even from the very first, and is characterized later by the presence in the vomitus of much blood (black vomit). **Hæmorrhages** from various other organs also are very common.

The mortality of various epidemics has varied from 15 per cent to 85 per cent. When severe, yellow fever is **one of the most fatal** of epidemic diseases.

The successful quarantine of a case of yellow fever is a matter of prime importance to the nurse, for the control, or lack of control, of the epidemic depends chiefly on her. The patient with yellow fever is not at all dangerous, nor can the Stegomyias in his room be dangerous until after twelve days have passed. The problem, therefore, is for her to prevent, by means of screens, all fresh mosquitoes from reaching her patient, and to kill every mosquito already in the house. If this is done, no one can "catch" yellow fever from that patient.

A fairly satisfactory specific anti-icteroides immune serum, obtained from immunized horses, is of value in the treatment

of yellow fever, provided it is given early enough, that is, during the first three days of the illness. The patient is to be made comfortable by cold baths, but great care must be taken that he does not catch cold. He must, because of the nephritis, lie perfectly quiet in bed. The question of diet is difficult because of the nephritis and also because of the vomiting, which often is so severe. The rule, therefore, is, no food but much water, and nutritive enemata if necessary. Various remedies, including cracked ice, may control the vomiting. Stimulants are often necessitated because of the profoundly collapsed condition of the patient.

Dengue is a pandemic fever of tropical and subtropical regions, including our Southern States, which is interesting because, during an epidemic, "there is no disease, not even influenza, which attacks so large a proportion of the population" (Osler), and also because sometimes it resembles yellow fever so closely that the nature of several epidemics has been in doubt.

This disease may be described, briefly, as a fever of brief duration (three days), with very sudden onset, which is accompanied by severe pain in the joints and muscles, with a skin rash which begins on the fifth day, which may be scarlatinal or macular in character, and with leucopenia (total count from 1000 to 5000 per cu. m.m.) and an increase of the small mononuclear leucocytes. The cause of this disease is not known, but is spread by a mosquito similar to that which conveys yellow fever. Because of its duration, some call it "three-day fever"; because of the severe, even exquisite, pains in the head, eyeballs, back, limbs, and in one, many, or all of the joints which accompanies it, it is called "break-bone fever"; because of the facial expression and the gait of its victims, due to the stiffness it causes in the muscles and joints, it is called also "pantomime fever," or "dandy fever." This disease is seldom fatal.

The treatment is that for any fever, with calomel for the bowels, large amounts of fluid, and aspirin or morphia for the pains.

Hydrophobia (rabies) is an acute, fatal, infectious disease, which may attack almost any animal and man. We are sure it is caused by a specific germ which, traveling along the nerves, finally localizes in the brain. Just what this germ is we do not know, but Negri's bodies, round objects about the size of a red blood corpuscle, are found so constantly in the infected tissues that their presence in the brain is considered sufficient for diagnosis. The disease is communicated from animal to animal or to man through the saliva of the infected animal. Dogs are very susceptible to it, but the bite of an infected cat is said to be even more dangerous. Only about 15 per cent of all persons actually bitten by mad dogs would, without treatment, contract the disease. The danger depends chiefly on the depth of the bite, and whether or not the animal's teeth penetrated first the clothes. The worst bites are those on the face and hands.

In man, the **period of incubation** of the disease is from two to eight weeks, seven being the average time. It is during this long period that we have a chance to prevent the development of hydrophobia.

After the period of incubation is over the disease develops in three stages. First, the **premonitory stage,** during which the flesh around the bite becomes inflamed and the patient mentally depressed and very irritable. Then follows the **stage of excitement,** which lasts from one to three days, during which even the slightest sensations produce violent spasms, especially of the throat and mouth. The attempt to swallow water produces spasms of the throat, and hence the patient writhes at the sight of it. The patients sometimes have maniacal attacks, during which they are dangerous, but these seldom develop. Next follows the **paralytic stage,** which lasts but for from six to eighteen hours and which terminates in coma and death.

The disease could be entirely eradicated if the laws to muzzle all dogs were rigidly enforced.

When a person is bitten by a dog suspected of being mad the animal should be at once killed and its **brain and spinal**

cord sent to the proper authorities. Smears of the fresh brain, which are dried in the air and then stained with a solution containing fuchsin and methylene blue, are then examined for Negri bodies.

The treatment of a bitten person should begin as soon as possible after the injury. Pasteur found that the virus of hydrophobia can, by repeated animal inoculations, be made so virulent that if injected into a rabbit the incubation period lasts, instead of fifteen or more days, only seven days. It is from the dried spinal cords of rabbits infected with this very virulent "seven-day virus" that the material used for inoculation is prepared. Subcutaneous injections of this are made **daily for twenty-one days,** thus establishing an early immunity. Then, when the virus which the dog's bite had injected into the patient has completed its incubation period of seven weeks, it finds the man already immune to this same disease. The results of this **Pasteur treatment,** when well given, are very satisfactory.

In all cases the bite **should early be cauterized** with carbolic acid or caustic soda, and the wound kept open. If the disease does develop, the outlook is hopeless. The patient is then kept as quiet as possible in a dark room, and morphia, chloroform, and other strong sedatives are used without stint. If the **spasms of the throat** cannot be relieved by cocaine the patient should be fed per rectum.

Rat bite fever, due, it is said, to Spirochæta morsus muris, is characterized by a **series of febrile paroxysms,** each lasting seven or eight days, and separated by one or two weeks of apyrexia. They may continue for months or years. The period of incubation may last even months. Then the original wound caused by the rat bite, and which perhaps has healed, becomes swollen, red, and ulcerated, and the regional lymph nodes enlarged. The attacks begin with a chill with a fever which lasts three or four days, then a scarlatinal or measley skin eruption appears and pain in the muscles and joints, and sometimes delirium develops. The wound should

be cauterized and the patient treated with arsphenamine or mercury.

Spirochætosis ictero-hæmorrhagica is another infection transmitted by rats, and due to Spirochæta ictero-hæmorrhagica, an organism present in the patient's blood during the first five or seven days of the disease, and in his urine for weeks later. The symptoms are acute fever with chills, headaches, general pains, vomiting, diarrhœa, and marked prostration. The jaundice (not always present) appears on about the fourth day and reaches its maximum on the tenth. Hæmorrhages from the mucous membranes and purpura also occur. The stools are usually clay colored. The liver, but not the spleen, is enlarged. The fever lasts about ten days. The mortality of this infection may reach even as high as 30 per cent. One keeps the patient in bed, on a liquid diet, and administers plenty of alkalies, salines for the bowels, and arsphenamine.

Filariasis is a disease due to a round worm, the embryos of which, Filaria Bancroftii, are found in the blood during the night. They are easily seen with a microscope since they are relatively large (about 1/75 of an inch long) and move very actively, driving the red corpuscles in all directions. **None are found during the day;** then they all are in the capillaries of the internal organs. A few appear towards evening, and **most are present at about midnight.** Another variety of this worm has embryos which are free in the blood during the day only, while those of a third variety are present both day and night. Their appearance seems to depend on the man's sleeping hours, for, if a day-laborer becomes a night-worker these embryos also change their hours.

This parasite is very common in British Guiana, but is found also in our Southern States. It is introduced into the blood of the victim by the **bite of some mosquito** which previously had bitten a patient with filariasis. The young embryo takes up its home in the lymph channels of the patient's pelvis, provided the mosquito's bite was on the leg and there grow to maturity, reaching a length of several inches. There

it **blocks the lymph vessels,** dams the lymph back into the skin of the legs, with **elephantiasis** as the result (that is, the leg becomes huge from the great thickening of the skin). Or, it may **block the lymph channels from the bladder,** which rupture pouring the lymph into the bladder. In such a case the patient's urine resembles milk **(chyluria)** or blood **(hæmaturia).** These adult worms produce the enormous number of young embryos which flood the blood, but which themselves do no harm, for they do not develop further.

The only possible treatment is to remove the adult worms by a surgical operation.

Trichiniasis is a disease of almost 2 per cent of the pigs in this country, that is, they are infected by the round worm Trichinella spiralis. In their muscles the little embryos of this worm lie well coiled up surrounded by a fibrous wall, which, just visible, look **like tiny grains of sand in the meat.** If such pork is eaten almost or quite raw these embryos will be set free by the gastric juice and during the next week will mature in the intestine of the man and the adult worms make their way into the mucous membrane. Here are born myriads of young, which are carried in the blood stream to all parts of the body. They settle in the patient's muscles, just as in the pigs, doing us no further harm. These embryos may be alive at the end of even twenty-five years.

It is while this multitude of embryos are being transported by the blood stream and are making their way into the muscles that the symptoms occur. The patient has a **fever** suggesting typhoid, **pain and soreness in all his muscles, œdema of the eyelids,** and a marked **eosinophilia.** A few of the patients die, some are invalids for years, but the majority show scarcely any symptoms at all.

The diagnosis of trichiniasis is suggested by the patient's admission that recently he had eaten raw pork, and by the symptoms. In order to be sure, however, one must remove a little piece of his muscle, usually from the calf, and there find the embryos themselves.

The treatment, successful only when the diagnosis is made

very early, is to purge the patient with calomel followed by Epsom salts, in order to get rid of the worms still in the intestines. After that there is nothing more which can be done.

Hookworm disease (ankylostomiasis and uncinariasis) is a disease caused by two quite similar little intestinal worms (Fig. 163, *C*) about half an inch long, Uncinaria americana

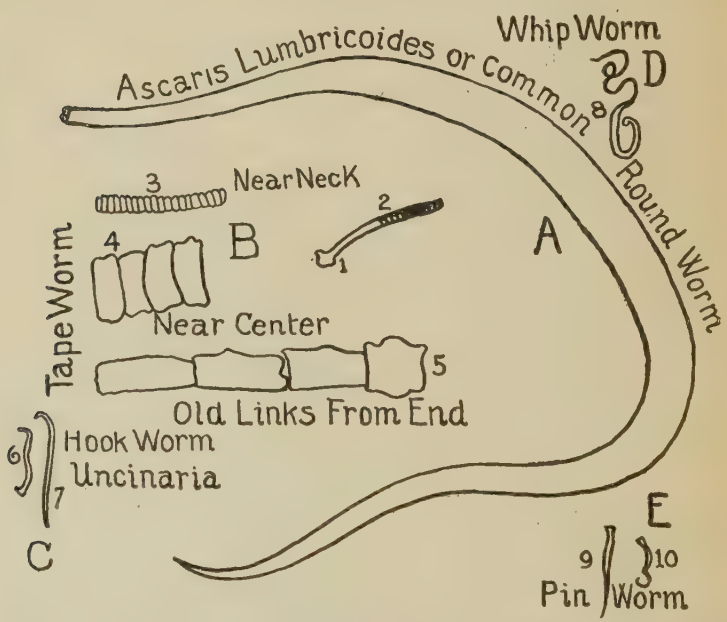


FIG. 163.—Some of the larger animal parasites. (All natural size.) *A*, ascaris lumbricoides or the round worm. *B*, fragment of a tapeworm. 1, the head; 2, the neck; 3, a fragment near the neck. *C*, the hookworm, the cause of the anæmia of the South. 6, male; 7, female. *D*, whipworm. *E*, pinworm; 9, female; 10, male.

and Ankylostoma duodenale, which live in great numbers in the upper part of the small intestine of the infected man. Their chief occupation seems to be to bite the mucous membrane in as many places as possible and thus cause a great deal of bleeding. The result of these minute hæmorrhages is a **severe secondary anæmia** which bears various names: hookworm disease, miners' anæmia, Egyptian chlorosis, tunnel-diggers' anæmia, brick-makers' anæmia, etc. It is one of

the most fatal of parasitic diseases. It is very common in our Southern States, where it is called "Southern anæmia," "anæmia of the poor whites," "sleeping sickness," etc. One jocose reporter named the worm the "germ of laziness" because easy fatiguability is so conspicuous a symptom of this disease. The embryos of this worm live in the dirt, sand, and clay, and easily infect a man either by mouth, if he eats with dirty hands, or by boring through the skin, causing, while doing so, "ground itch." The patient is anæmic, and, if a child, develops to maturity slowly; the skin has a muddy, pale, hue ("Florida complexion"); the eyes are dull and heavy ("fish eyes"). Later the skin is œdematous and the patient very weak and short of breath. Many patients, if not treated, die.

The diagnosis of hookworm disease is easily made since the **eggs of these parasites are abundant in the stools.** The worms are easily killed by a few doses of **thymol.** To accomplish this, the patient, without supper, is given a saline purge at night, and early the next morning three doses one hour apart of thymol, 15 grains each, coarsely powdered and in capsules. Two hours later another saline purge is given to remove the thymol.

Ascaris lumbricoides, round worm, is a very common intestinal parasite (Fig. 163, *A*) of children especially, which is from four to twelve inches long and about one eighth of an inch in thickness. It lives in the small bowel and, so long as it stays there, seems to do no harm. But **when it wanders,** as it does if the child contracts any **fever,** it may do serious injury. Sometimes it creeps into the stomach and is vomited; sometimes it gets into the throat and then into the trachea; from the throat it may force its way into the middle ear by crawling through the Eustachian tube, rupture the drum, and appear in the external ear; it may wander up into the common bile-duct; it may thrust its head through a gastric or typhoid ulcer, causing peritonitis.

Many symptoms of children—nervousness, twitching, con-

vulsions, etc.,—are attributed to these worms, but are probably due to other causes.

These worms are easily removed by a dose of **santonin.** Santonin, 0.30 gms., calomel, 0.20 gms., and milk sugar, 3. gms., is divided into 10 powders. One of these powders is taken each hour for three doses, and then a dose of calomel given. Or, better, 1 c.c. of oil of chenopodium (wormseed oil) is swallowed on an empty stomach, and followed, in one hour, by a purge of Epsom salts. This treatment should not be early repeated.

Pinworm (Oxyuris vermicularis) the "threadworm," or "seatworm," is very commonly found in the rectum, especially of children. This is a small, white, thread-like worm, about a quarter of an inch long. The chief symptom it causes is **intense itching,** especially at night. Santonin may expel these worms, but local rectal irrigations of strong salt solution, or of quassia water, are most effective (Fig. 163, *E*).

The tapeworms are large and long flat worms that live in the intestines of men. They consist of many thin, broad segments or 'links" (Fig. 163, *B*).

The beef tapeworm is the most common tapeworm in America. One gets it from eating the insufficiently cooked meat of an animal in whose muscles were the embryo of this worm. In the bowel of man this worm grows to a length of from fifteen to twenty feet. Its largest links are nearly a quarter of an inch broad, and about half an inch or more long. The head is about the size of a common black-headed pin.

The pork tapeworm is very rare in America. It develops from a living embryo of this worm—eaten with infected raw pork. It is smaller than the worm described above, being only from six to twelve feet long, and has somewhat smaller links. Its head also is smaller, but is provided not only with suckers, as is the beef worm, but also with **hooks.** It is a much harder worm to expel than is the beef worm.

Bothriocephalus latus, a tapeworm common in parts of Europe and the far East, and very rare in America, is gained

by eating raw fish. It may grow to a length of from twenty-five to thirty feet. This worm sometimes causes an anæmia which can hardly be told from primary pernicious anæmia.

The symptoms caused by the beef and pork worms are few, if any, until the patient finds out that he has the worm, and then any number of most distressing nervous symptoms may develop.

The diagnosis of all large tapeworms is easy, for their **links appear in nearly every stool of the patient,** and, when seen, cannot be mistaken. But the opposite mistake is often made, for many patients are certain that the shreds of mucus or of vegetable fibres that they pass in their stools are tapeworm links, and they explain the ragged translucent appearance of the "worm" by the supposition that it is "decayed." The segments of a tapeworm, however, do not easily decay.

One avoids tapeworms by abstaining from little cooked beef and pork. In America the meat inspection must become much more rigid before it will be safe to eat the raw meat which some enjoy.

The treatment of tapeworm consists in first cleaning out the bowels well with saline purges, and then giving a large dose of one of several drugs. This dose should at least so stupefy the worm that it will cease to cling to the intestinal wall and so will be passed in the stools. Among the remedies used are **male fern,** pomegranate root, turpentine, and (one of the best for children) **a pumpkin-seed tea** consisting of water in which three or four ounces of crushed pumpkin seeds have been soaked (but not boiled) for twelve or fourteen hours. A purge must be given soon after one of these remedies has been administered, in order to make sure that the worm is hurried out of the bowel. The night before the treatment one cup of soup only, or one glass of milk, is allowed, then a mild laxative or enema. The next morning only one cup of coffee or tea is permitted. Half an hour later the patient is given the vermifuge. For adults we use the oleoresin of male fern, given in 2 grms. capsules, with half a glass of hot water, and a smaller dose one hour later. Two hours

after the second dose the patient is given an ounce of Epsom salts (not castor oil). The patient should lie in bed during this treatment. One must always **find the head** of the worm before he can be sure that the cure is accomplished, for, even though twenty feet of worm are passed, if the head is left behind there will in a few months grow from it twenty feet more. The head is easily recognized, for it is a small ball, about the size of the head of a pin, on a thread-like neck. The stool which is expected to contain the worm should be passed into a vessel nearly full of warm water. If the water is cold or if cold air strikes the worm it may vigorously contract and break, and the head be left in the bowel.

Cysticercus disease is an infection of the body by the pork tapeworm (page 543). If we eat pork containing an encysted embryo of this tapeworm, this embryo will develop in the patient's bowel into an adult worm. If, however, instead of an embryo we **swallow an egg of this tapeworm** this will develop in the bowel into an embryo which will make its way through the wall of the stomach or intestine and be carried by the blood to almost any organ of the body. Wherever one of these larvæ settles it soon builds a wall around itself, thus forming a **small cyst,** from one-quarter to three-quarters of an inch long and about half an inch in diameter, which contains the head of a tapeworm. This develops no further and grows no larger. Muscles containing these cysts are said to be "measled." The muscles, internal organs, and skin of an infected person may contain thousands of these little cysts. The most of them do no harm, but should one locate in the brain or the eye, etc., it can prove very serious, as would any tumor of that size in that location.

Hydatid disease is due to a small tapeworm, Tænia echinococcus, which when full grown is only about one-fifth of an inch long, which inhabits the intestines of dogs, and which in man can cause far more serious disease than any of the larger worms. If an egg of this worm reaches the stomach it hatches, and a tiny embryo is set free which burrows its way through the intestinal wall and which is then carried by

the blood stream to some distant organ. Wherever it lodges it forms **a little cyst, or bladder,** which contains the head of the worm. Thus far it resembles the cysticercus of the pork tapeworm, but its later development is very different. This little bladder can **bud and bud again,** hundreds of times, each bud producing a new cyst similar to and as large as the first. These "daughter cysts," in turn, may bud and soon contain many "granddaughter cysts." The one original cyst, now of great size, may contain hundreds of these small cysts.

The symptoms of one of these cysts are those of a tumor and will depend on the organ in which it lies. Echinococcus disease is common in Australia, Iceland, and parts of Europe, —wherever dogs are allowed to live intimately with men. It is rare in America.

The treatment is to remove the cyst by surgery and with great care lest it rupture during the operation.

CHAPTER XXIV

The Psychoneuroses

Thirty years ago a large percentage of the patients in the medical wards of our hospitals were diagnosed as cases of **neurasthenia.** Neurasthenia was then described as a state of nervous exhaustion which presented also some symptoms suggesting organic disease. That is, two elements were recognized; the easy fatigability, and the symptoms suggesting diseases which could not then be demonstrated.

Now, a diagnosis of neurasthenia is seldom, by some is never, made. We recognize that many persons are by nature "neurasthenic," by which we mean that it is their mental habit to be introspective, to worry easily, and to suffer acutely from trivial causes, but now if we find that their health until their present illness had been good and if we can exclude psychasthenia (page 552), which was the correct diagnosis of many of the neurasthenics of that day, instead of stopping with the diagnosis "neurasthenia" we search carefully for some physical disorder. Under what heading do we now catalogue those patients who, thirty years ago, were card-indexed under the title neurasthenia?

Before beginning this discussion we should bear in mind that then we had no efficient X-ray apparatus, no methods of serology, no knowledge of basal metabolism, no blood chemistry, little blood bacteriology, no apparatus to measure blood pressure, few of the satisfactory tests for examining the spinal fluid, no methods for the functional diagnosis of any organ and little appreciation of the milder grades of mental diseases. In those days if the methods of diagnosis at our disposal failed to reveal some definite lesion the patient was pronounced "free from organic disease"; and if that statement failed to relieve his suffering, he was said to have "neurasthenia." Now we know that the **great majority of those patients must have had slight grades, or early stage, of organic diseases;**

thyroid troubles, tuberculosis, heart disease, septicæmia, nephritis, cancer, lues, paresis or mental diseases, etc. True, the correct diagnosis of many of those cases of neurasthenia did later become only too evident, but others got well without a better diagnosis.

The correct early diagnosis of these cases is very important since the cure of an organic disease is easy the earlier it is made. Unfortunately many are incurable when far enough advanced to be easily recognized. Of course this does not entirely dismiss the problem of neurasthenia. The neurasthenic person is still a problem, and any normal person can become in some degree neurasthenic. But if this habit of mind is a recent development, if it was not evident at puberty, then that patient probably has some organic trouble for us to find, and he should not suffer the neglect which tends to arise if we recognize merely his attitude towards his symptoms, and, therefore, dismiss him as a case of neurasthenia.

In order to understand the following paragraphs a few general statements may be in place. Of **all the sensory nervous currents** which travel from different parts of our body to our spinal cord **few give rise to conscious sensations.** The pulsations of our heart are very powerful, and the heart makes considerable noise every time it beats; our stomach empties itself by means of vigorous peristaltic movements; the wonder is that we are not in actual pain all the time. But these sensations normally are "subliminal." Let *c-d* (Fig. 164) be the limen, or threshold, of consciousness. We may suppose that only those sensations which the mechanism of our nervous system allows to rise above this line are noticed. That is, we should be unconscious of *g* and just feel *e*, but *f* would probably cause a severe pain. The great majority of these stimuli which must arise from the normal functioning of our organs are subliminal even though vigorous. Other groups of stimuli, touch, sight, hearing, smell, etc., which we may assume to be much weaker, register with intense force on consciousness. So, normally, we do not hear our heart beat, feel our stomach contact, etc. But this limen has no constant

level; we are continually raising or lowering it. When we are interested in a book, for illustration, we do not hear our friend speak to us; when we are walking absorbed in thought we may unconsciously pass a friend without seeing him. The limen for these sensations has temporarily been raised. Again, when we are intensely in earnest we strain the eye to see, or the ear to hear, or the fingers to feel that which interests us, and by so doing we lower the limen to these sensations, so that stimuli which under ordinary conditions would be subliminal are perceived. This is what neurasthenic persons

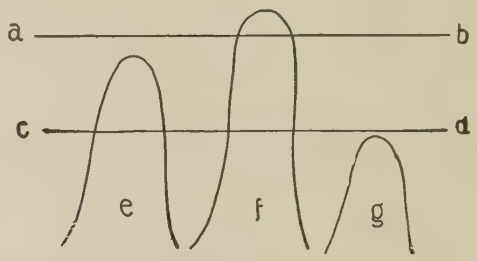


FIG. 164.—A diagram of our sensations. *a-b,* the limen of consciousness when we are not paying strict attention to our sensations; *c-d,* the limen when we are paying strict attention to them. The sensation, *e,* will be perceived if the limen is *c-d,* and not noticed if it is *a-b.* The sensation, *f,* will be just perceived if the limen is *a-b,* and will be painful if it is *c-d.* The sensation, *g,* will escape notice even by the person whose limen is *c-d.* Most of the stimuli to the brain are like *g.*

in particular are in great danger of doing. They seemed to have lowered the limen of consciousness for sensations from an organ which worries them, and so they feel many more sensations, and all of them acutely, than would the normal man. This man hears of, or reads of, or his guilty conscience suggests, a disease of a certain organ, and he begins to study his sensations to see if perchance he may have any of its symptoms. Too often, however, the organ he worries about is not the one which is diseased. In fact, certain organs seem to act as fire bells and give the alarm for latent exhausting disease elsewhere in the body. For illustration, of each twenty persons whose symptoms are gastric not over one will have a stomach disease. In the other nineteen cases to treat the stomach for the stomach symptoms would be

comparable to pouring the water on the fire bell because it is making all the noise. This is why the diagnosis of these conditions requires such care.

There is also a surprising **lack of relation between the seriousness of a disease and the severity of its symptoms.** Nature is kindly, and the toxins of serious disease often have an anæsthetic effect. Very few persons suffer much while acutely ill. This surprises the inexperienced hospital visitors who expect disease and suffering to be correlated. They look down the rows of ward beds containing apparently comfortable persons and ask, "Is anyone here really ill?" Some patients are partially anæsthetized, "toxic," during the entire course of a fever, while most very ill patients gradually become dull and then unconscious as death draws near. Typhoid fever, for illustration, is, after the first week a relatively "comfortable" disease. This makes difficult the early diagnosis of a peritonitis from the perforation of a typhoid ulcer (page 406). In fact, surprisingly few very ill persons really have much physical pain. Of course the problem is quite different with most surgical patients and with patients who have any of the various colics—gall-stone, renal, etc., for these belong really in the "traumatic" group.

But the neurasthenic person suffers acutely. He gets angry when truthfully assured that the "terrible pain" around his heart does not denote heart disease, and yet in real heart disease the heart itself seldom gives pain. He cannot understand why the "unbearable pains" in his back do not indicate Bright's disease, although Bright's disease is practically always painless. His pains are "terrible" and "unbearable," as some prove by their suicide. For just that reason we may overlook the organic disease which too often is there. This is well illustrated almost daily at the entrance door of any large hospital. "Doctor, come at once. The man is suffering terribly. He is screaming with pain," telephones the clerk who admits new cases, and the doctor may finish what is then occupying him, muttering to himself, "neurasthenic probably." "New patient," says this same attendant on another

occasion, "but he seems very comfortable"; and the experienced interne hurries, for he knows how many of those "comfortable" patients are very ill. This neurasthenic habit of mind should be understood by the nurse. It leads the patient to magnify with the lens of the microscope of the mind the slight symptoms. The microscope does not, however, create the object whose image it enlarges. And so it is with the disease of the neurasthenic. It too often is there and should not be overlooked merely because its symptoms or, what is more often the case, the symptoms of the wrong organ, are so magnified.

But the important element in neurasthenia is not the suffering but the fatiguability of the patient. Nervous force is somewhat like money. In youth we have a certain reserve capital of this force, and by rest, sleep, and food we earn a certain daily income, much of which we daily expend. Those whose reserves are large, whose income is good, or whose expenses are small, seldom suffer. But those whose income is small, or who are extravagant in their use of it, sooner or later come to nervous bankruptcy. Those who inherit a "nervous disposition" (which usually means extravagant mental habits), but also those who eat poorly, sleep poorly, work hard, and, above all else, those who worry, sooner or later come to grief. The problem is not how hard a man works, or how important or how pressing his work is; the question is How much nervous force does he expend in doing it? Some persons in performing a simple task would seem to spend twice as much nervous force as others. Worry, however, is the usual explanation of an excessive expenditure; worry is the "friction" in the mental mechanism. Also, we may with good reason liken our nervous system to a storage battery. Of all the "leaks" of nervous force, of all the "short circuits" of these batteries, worry is the worst. Unfortunately the body does not provide a very successful "antitode" for nervous exhaustion; this is not, like fevers, a "self-limiting disease." True, changes in the emotions of the fatigued, as inability to concentrate, loss of interest, depression, or even

antipathy to his work, would seem to be protective mechanisms to slow up our expenditures of force and to save us, but usually these become parts of a vicious circle, for the neurasthenic tends to sleep little, to eat less and less, and to worry more and more about his condition, all of which means, sooner or later, great mental fatigue. As a result he cannot apply himself to mental work; he is "too tired to think." If he is a business man, he may become incapable of decision, anxious, and "absent-minded"; others, egotistical, irritable, and extremely uncomfortable to deal with; all become very emotional. This is the experience of many a normal man who overworks.

The neurasthenic needs, first of all, an accurate diagnosis. There always is **some reason for the easy fatiguability of one previously well.** It may be uncorrected eye strain, infected tonsils, teeth or nose, it may be an early or slight disease of the lungs, heart, gastrointestinal canal, central nervous system, or glands of internal secretion, it may be flat feet, it may be a cancer hidden away in some organ. To find it is our first problem, to cure it, if possible, our second, and to reëducate the patient by proper psychotherapy, our third. This will be described on page 554.

Psychasthenia is a quite different matter. This is a term applied to a mental pattern demonstrable in the individual from childhood on, which allows him to be dominated by fears and easily to develop obsessions which may make life miserable. Psychasthenia is more than a habit, it is a **constitutional peculiarity.** To justify this diagnosis the patient's history should show that psychasthenic traits were present as early at least as puberty. The natural result of years dominated by fears against which, naturally, these persons fight, is exhaustion, that is, asthenia, which often is called neurasthenia. These patients, like others, develop organic diseases, but they do not become psychasthenic or develop psychasthenia, they would seem to be born that way. Cure it we cannot; teach them how to control it, we can and should. Psychasthenia does not shorten a man's life; in fact, it

lengthens it, for no one takes better care of himself or demands better care from others than does he. He never dies from his malady, but his whole family may be literally sacrificed to it, for he sometimes demands their care and consideration with a jealousy which almost amounts to an insanity (although these patients themselves seldom become insane). Hence it is that every psychasthenic successfully educated is a family released. Treat one hundred persons ill with typhoid fever by simple nursing only, and *vis medicatrix naturæ* will cure at least eighty of them. For these eighty cures no doctor can claim credit. For every psychasthenic relieved, however, some one deserves a great deal of credit. Typhoid incapacitates a man for but a few weeks; psychasthenia usually for a lifetime. The typhoid patient dies but once, the psychasthenics "die daily." They suffer with an intensity actually not equalled in organic disease and they make their families suffer with them.

Psychasthenia may not for years interfere with a patient's success, but, as a rule, sooner or later it leads to nervous bankruptcy. Then it is that his fears or "phobias," ever present but until now held under control, get the better of him. Some actually cannot cross an open square (agarophobia), some cannot remain in a small room (claustrophobia), some fear the dark (nyctophobia), some cannot bear to be alone (monophobia). Some fear dirt (mysophobia), some fear diseases (nosophobia), as tuberculosis (phthisiophobia). Some no longer can control their formerly suppressed superstitions, and now their fears for the figure 13, the left hand, Friday, etc., may make the day's work impossible. These patients suffer also certain physical symptoms; headaches on the top of the head, pain at the back of the neck and at the lower end of the spine, muscular weakness, dizziness, a rapid irregular heart, a painfully throbbing aorta, nausea, vomiting, diarrhea, skin flushing and itching, and a host of other symptoms.

The diagnosis of psychasthenia will rest partly on the **patient's family history,** since this is a definitely inheritable condition, more on his **own personal history,** since we should

hesitate to make this diagnosis unless we get evidence of **its presence at or near puberty,** on the physical symptoms mentioned above, and on our ability to exclude true diseases, which it is always difficult to do.

The treatment of psychasthenia, and for all the neurasthenics who in addition to their organic diseases have mental symptoms which need therapy, is largely a problem for the nurse and deserves her most careful study. This is, in fact, the highest test of a nurse's ability. While each patient must be treated individually, there are several general rules which hold for all. The treatment should be carefully planned and this plan followed in every detail, else little will be gained. To carry it out 100 per cent well may mean success, while if only 90 per cent well may mean failure as surely as if only 10 per cent well. First, the patient should be treated in a hospital or sanitorium. One may utterly fail at home, where every familiar sight and sound suggests to the patient though in bed, the factors which led to his present condition. It is essential that he **relax,** that he gain **rest of mind and body** and this can be done only if we protect him from all possible reminders of the pains and even of the pleasures of his life. Second, it is essential that the patient have **full confidence in the nurse's ability to help.** A nurse whom the patient can persuade to yield in even a minor point in the doctor's plan, or who shows herself uncertain or ignorant of any of its details, would seem to arouse in the patient an emotion akin to that which a drowning person feels who clutches frantically at a floating object only to find that it sinks with him. But if it supports him, then he clings to it with desperation and satisfaction, little caring what it is. So the psychasthenic clings to that nurse who shows herself able to help.

Third, the major problem is a **real good rest;** a physical upbuilding. The patient is a nervous bankrupt. He must first relax, then rest, sleep, and eat. We talk little at this period about the future. Certainly no scolding, no advice even are allowed, no plans are discussed. Now he must rest. It is interesting to observe how quickly a patient, far away

from home, in a hospital room, in bed, all business ties cut off, and under the care of a suitable nurse who inspires his confidence, becomes relaxed and drowsy. The sensations of this relaxation are so definite that they may even frighten him. Some patients are sure they are seriously ill; some that they are dying. They are like the man who while his house was on fire could move all the furniture with "superhuman" strength, but now, after the fire is out, he discovers for the first time his burns and bruises, and even faints.

Since the fatigue—physical and mental—is usually profound, the patient should **spend at least three or four weeks in bed.** We may promise him that the more nearly horizontal he keeps his body, the less he sits up—even on one elbow—the more relaxed all his muscles are, the sooner he will be up. It is well to keep the bed outdoors during the day and if possible also during the night.

Insomnia is one of the important symptoms to combat, but it should not be met, except possibly for the first week, by drugs. The cold pack, massage, the warm bath, and a cup of hot milk in the evening will help the patient to sleep. He will in time sleep well without such aid.

These patients **should be overfed.** One does well to commence, as Dubois advises, with a week of pure milk diet, the milk each two hours, two ounces each time at first and gradually increasing it until he is receiving three, four, or even five quarts a day, and then, on the seventh day, abruptly (and unexpectedly to the patient) changing to a mixed diet of even surprising abundance. The chances are that our patient has suffered from a few or many of the gastric symptoms described under nervous dyspepsia; the chances are that his doctors have limited his diet in various ways, one of them cutting out "acid foods," another starches, another, fats, etc., until he has come to the hospital in a half-starved condition and is afraid to eat almost everything. If our diagnosis of psychasthenia is correct the stomach, however much pain it may have caused, can and should digest painlessly almost anything within reason, and will do so

if it is made to. The stomach is like a spoiled child; the more it is considered, the more unruly it becomes; the more attention it gets, the more pain it gives. It needs, figuratively, to be "made to mind." One does well to prescribe the foods as he does therapies. The patient is told that everything on the tray is medicine to be eaten to the last crumb and drunk to the last drop. Some patients who suffer much from a certain dish when eaten as food will not suffer from it at all when it is eaten as medicine. The patient should gain weight. The gain itself is unimportant, although it usually is a reason for encouragement, but there is little doubt that nervous rehabilitation is most successfully accomplished during periods of overnourishment.

Hydrotherapy is a very valuable therapy. The cold pack at night and the cold sponge bath in the morning are among the best tonics of the nervous system, although some patients sleep better after a warm bath.

Many patients are **constipated,** and this **should be overcome through habit and diet.** Granting that there is no disease of the appendix or gall-bladder and no rectal trouble, which, if present, should be cared for at the beginning of the treatment, the patient should attempt to have the bowels move at the same hour of each day. To accomplish this the diet should be rich in foods containing cellulose and fruit acids and sugars, such as fruits, cereals and green vegetables. The breakfast should contain a fruit, a cereal, and honey, marmalade, or jam. A patient who had taken a laxative daily for twelve years was able in three weeks to form so fixed a habit that two years later he said "he did not need to look at his watch to see when nine o'clock came."

Psychotherapy is the important element in the treatment of psychasthenia. By this we do not mean a lot of "jollying," or mere "cheering up," but carefully studied conversation supplemented later by carefully chosen reading. The patient should understand his condition and be taught how to control his symptoms. No scoldings, only encouragement; no lectures, only carefully planned hints and suggestions fre-

quently repeated, should be the plan. Finally, the success of the doctor depends in large degree on the support he receives from the nurses.

After resting in bed for the prescribed time the patient uses a back rest, then sits up in a chair, and then walks. At this point begins the most important part of the treatment, for all of his previous symptoms may now return temporarily and with increased violence. **The program for the day should be written out,** something to do each hour—reading, walking, resting, and working. The amount of exercise should increase daily. Now is the time to reëducate him, to show him that the things which formerly he thought he could not do, he now can. Tell him that he was like an electric automobile run down. Its recharging is a slow process, and while this is going on there is little to show, but when the machine is put in motion then the force is evident. Next follows a vacation, away from home and away from the hospital. Then home and work, with renewed strength, energy, and enthusiasm, and with the recently acquired habits so strong that he will not soon relapse.

No "cure" of a congenital psychasthenia is possible. The problem is to help him to avoid the mental fatigue on which his breakdowns depend. He should at regular intervals take a vacation and will always need encouragement. He has been likened to an eight-day clock which must be wound up regularly, at such intervals as experience with his case has indicated as best.

Hysteria also is a congenital condition, but one which, so far as the family and society are concerned, is of much more serious character than psychasthenia, since the patients with hysteria exert, because of the nature of their trouble, a much more positive influence. The psychasthenic impresses those about him as being a weakling; the patient with hysteria dominates his environment.

Hysteria and "hysterical condition" are not the same. Any unstable emotional person with poor self-control might be called "hysterical," while the worst cases of true hysteria

are just the reverse. They outwardly are calm, self-controlled and serious. They are not malingerers, but believe in themselves implicitly. None of our patients is more sincere. There is the danger. He is the helpless victim of his own suggestions. While this condition is most common in women, it occurs in men also, and it spares no age, that of children under puberty being the hardest to detect. **The first evidences of hysteria,** although not often then recognized, **appear at or before puberty.** Given a negative history of this period, and one should be very cautious in venturing this diagnosis in the case of an adult.

Hysteria has been defined as a disease in which the mind produces some of the objective signs as well as the subjective symptoms of organic diseases. A person with hysteria unconsciously may present the picture of almost any disease, especially a serious one, with practically all of its signs and symptoms almost exactly reproduced, but with, in addition to these, certain signs which are peculiar to hysteria, and which are called the **stigmata** of this disease. Among the stigmata mentioned in the history of the case may be a history of a period of true **aphonia,** or inability to speak (they whisper clearly, but make no vocal sound, not even the hoarse growls which cases of non-hysterical aphonia due to disease of the larynx can make); or, a history of a period of **temporary blindness**; the record of **paralysis of any muscle**; and of **convulsions** not definitely epileptic. Among the stigmata of the present illness are: sighing respiration—that is, a rhythmical "catching of the breath" every fifth or sixth respiration; the "globus hystericus," that is, the sensation of a ball rising in the throat; areas of anæsthesia of the skin, usually between the nipples; contraction of the fields of vision; a reversal of the fields of color vision; a tendency to emotional outbreaks; convulsive seizures of various kinds; temporary paralyses; a surprising lack of appreciation of the possible importance of their apparently serious condition (for illustration, they view with interest, not concern, the details of their convulsions, and they view with apparent pride a paralysis

which suggests possibilities that would terrify a more normally minded man); and, finally, syndromes of serious organic conditions but which always present certain definite discrepancies in the physical signs.

A broad **general classification** of the types of hysteria separates the **convulsive from the non-convulsive forms. The minor convulsive form** may be illustrated by the attacks of uncontrolled and alternating laughing and crying which the lay mind associates with "hysteria." The best illustration of the major convulsive form is the convulsions which may resemble epilepsy so closely that only an expert can detect the differences. They do, however, differ. **During the convulsion of hysteria** the patient may hurt others but never himself. He always falls safely, never "hard," as if shot. He never chews his tongue, and his pupils, during the entire convulsion, will react to light. That he is not for one moment truly comatose is evident from his continuous muscular resistance to the efforts of the person trying to control him. The true epileptic is truly and completely unconscious and helpless, but "it takes six men" to hold on the bed the patient with a convulsion of major hysteria.

Among the **non-convulsive forms** are the hysterical paralyses. There is no type or form of organic paralysis which may not be simulated in hysteria; hemiplegia, paraplegia, monoplegia, all occur. Of course, between these and the paralysis due to organic disease there are differences, especially in the distribution of the paralysis and in the deep reflexes, and certainly in the course of the disease, since the case of hysterical paralysis suddenly and "miraculously" gets well. But his muscles are, for the time being, as truly paralyzed in the hysterical as in the organic type,—that is, the will is unable to contract them. We have seen patients who have been helplessly paralyzed for six years get up and walk. If this were to happen in a religious gathering it would be a miracle, but since it happened in a clinic it is merely the successful (and temporary) relief of one hysterical functional disability.

Other non-convulsive forms mimic many of the incurable

diseases of the spinal cord—lateral sclerosis, insular sclerosis, etc. Some patients present various types of rhythmical spasm, of tremors, etc., which may continue for months. Still others have areas anæsthetic to touch, pain, etc. Others, on the contrary, are exquisitely hypersensitive over certain regions. Some hysterical patients for months are blind, others are deaf. They are honest in their statements, for they may be psychically blind, psychically deaf, or anæsthetic. The fact that during these periods we can prove that the sense of vision, or of hearing, does determine their reflex and automatic actions does not disprove their statements that they (consciously) do not see, do not hear, or do not feel. Among their hysterical pains, which would seem to be severe, are headaches, especially on the vertex of the skull, backaches at the nape of the neck and at the coccyx, pains in the gastric region simulating those of gastric ulcer, pains in the appendix region, and, especially, pains in the pelvis. Certain gastrointestinal features of hysteria must be very distressing: pains, nausea, vomiting, constipation, violent diarrhœa, and, finally, not only loss of appetite, but so positive an **aversion to food** that extreme emaciation and even death result (anorexia nervosa).

Some persons with true hysteria have **dyspnœa**; some a hiccough which may last months; some have **spasm of the respiratory muscles** which produces peculiar cries, whoops, or noises that resemble those of animals.

Finally, the literature of hysteria reports cases of "hysterical arthritis," "hysterical fever," etc. It should be remembered, however, that the most of these cases were reported before the days of our modern methods of diagnosis, and that, therefore, the problem of the diagnosis of hysteria resembles much that of neurasthenia (page 547). Certainly there is no reason why a case of true hysteria should not contract tuberculosis, acute arthritis, acute appendicitis, etc. Unfortunately the knowledge that a person has psychasthenia or hysteria has led to great injustice in his medical care.

When passing judgment on cases of hysteria one is in

danger of being very unkind. These patients believe in their diseases more firmly than do their friends. They usually are "unfortunate in their parents" since they have inherited strong neuropathic tendencies; as a rule they have been unfortunate in their early training, in that they have never been taught self-control or the rights of others; and, unfortunately too, many of them have been under the care of doctors who have misunderstood their condition.

The treatment of hysteria is in many ways like that for psychasthenia, only more vigorously carried out. The isolation of these patients should be complete so far as the immediate relatives are concerned. Do this, and it is surprising how soon the convulsions, rhythmical spasms, etc., present for weeks or months, disappear. Emphasis should be laid on the re-education. Here the trained nurse well trained for this work is indispensable. The patient must be handled with tact and firmness. He must be taught how to control himself. His life and work must be mapped out for him. His friends should understand him.

Many specialists believe that hysteria is a congenital defect of such gravity that persons thus afflicted should early be declared wards of the State and not be allowed to marry, since their children are so apt to be in some way defective. Certainly persons with hysteria have been among the greatest of trouble-makers, especially in the home and in the church. Their religious experiences, like their paralysis, are so spectacular, so bizarre, both in their origin and termination, that they cannot but deceive those with a will to believe.

In addition to the above-described so-called functional neuroses there are **two mental conditions,** the manic depressive psychosis, and dementia præcox, which in their severer forms are true insanities and therefore of no interest to us now, but which in their milder forms are often mistaken as "functional," harmless psychoneuroses.

Insanity

At this point we would state that "insanity" is a legal, not a medical, word, and denotes a condition which renders

the patient such that it is no injustice to deprive him of his personal liberty, or of the control of his property, or of both. In other words, the person judged insane is considered definitely irresponsible and is, therefore, both for his and for the public's sake, confined in a closed institution. The diagnosis of insanity is not made by doctors. They may give their testimony before the proper court, but it is some judicial authority which commits the case to a hospital for the insane, and if, later, doubt arises as to the justice of the commitment the question will be decided, not by doctors, but by a judge and twelve jurors who too often treat the doctors' opinions with very little respect. **Insane conditions,** however, are merely **severer grades of certain mental disturbances** the milder grades of which pass as "nervousness," "neurasthenia," "nervous prostration," etc. Some of these mild cases of mental disease run their course and get well without ever having aroused on the part of wife or business associates the question of a mental disease. Some cases which begin mild and are treated for "nervousness" later progress to a degree of undoubted "insanity." The accurate early diagnosis of these so-called neuropsychoses is a matter of great importance to the patient and his family. Some may be cured without the stigma of commitment; others may be committed before they have disgraced the family. In the diagnosis of all such cases the accurate observation and notes of the nurse are very valuable indeed to the doctor.

It is of importance that cases with the severer grades of these two diseases, manic-depressive psychosis, and the later stages of dementia præcox, together with the cases dementia paralytica (Page 531) explain about eighty per cent of the population of our insane hospitals.

Manic-depressive psychosis explains about one-third of the above-mentioned group. In its marked stages there is no doubt as to the diagnosis, for the patient is either "wildly maniacal" or definitely "melancholic" and perhaps suicidal. The mild stages, however, are very deceptive. The typical case of manic-depressive psychosis presents a sequence of

mild exaltation with some instability, lasting for a few days or weeks, followed by a period of depression which may last for months. We even can draw a chart of these phases and from its shape can roughly predict the future progress of the case. In more of the mild cases, however, this circular course is not definite, and one must use some imagination to identify the phase of exaltation. A marked feature of this psychosis is its tendency to recur, sometimes with great regularity, at periods of from three to ten years. Some persons of great genius and ability have been subject to such periods of depression.

The patient describes **the mild manic phase** as a period of ambitious striving, of restless effort, of exaltation, of insomnia. His imagination is lively, his self-control poor. He attempts far too much, and yet may be conscious that he is not as efficient as he should be. He is talkative, his conversation a stream of ideas showing very little logical sequence. One word in a sentence will suggest a new and unrelated turn of the conversation. This phase may be brief, lasting only a few days or weeks, and then follows the **period of depression.** The patient becomes unhappy, depressed, despondent; life seems to have lost its value; he accuses himself of inefficiency; of failure. The mild case may be unhappy **during the early morning hours only,** while during the afternoon of the same day he feels quite normal. Those cases a little more severe than the above are depressed during the whole day, but usually more so in the morning. A mild case may continue successfully at business, he and his family interpreting his condition as tired out, discouraged, the "result of overwork." He talks of the need of a vacation. As a matter of fact, however, he has a strong feeling of his own unworthiness, of disappointment at his supposed failures and imagined shortcomings. He repents of the sins of his youth; he may become very religious. Some, during a stage of depression, have changed their business and become religious workers, just as others during the period of excitement have

indulged in unaccustomed debauchery. Let the depression become a little deeper and suicide is a logical act.

Of course many persons of keen mentality, many of them "geniuses," have their periods of discouragement and "depression" but it is not of this type. Their "blues" last but for a few hours or days; there is some good reason why they should just then feel depressed, and good news will at once relieve them. On a vacation, and especially if they visit a congenial place, this depression is very quickly dispelled. But not so with the manic-depressive case. One depressed day follows another with **monotonous regularity.** Do what he will, go where he may, be as successful as he may desire, still the depression continues, perhaps at times temporarily less, but still it is there. While the succeeding days may differ somewhat yet there is a **definite progress in the condition.** That is, at first over a period of weeks it gradually becomes more and more marked, and later less and less so until the patient is normal. In some cases, however, the condition does clear up suddenly, much to the delight of that quack medicine dealer or miracle worker who last had this patient under his care.

Still deeper depression makes the patient confused, "doesn't understand what it is all about," becomes very suspicious of his surroundings, refuses to eat, fears poison in his food, and often is hard to control. Then commitment to a closed institution is necessary. In these later stages, however, we are not now interested. We speak only of those cases so mild that even the wife believes her husband only "tired out."

The diagnosis of this condition is very important since even the life of the patient may be at stake. First, the patient, if an adult, will give the history of **several misunderstood attacks,** perhaps at regular periods of from seven to ten years, and each of increasing severity. The first is often interpreted as a brief episode of puberty; the second, often while the youth is in college, may have led him to be very serious, and perhaps to choose unwisely his future career;

the third, a few years later, is often called "nervous prostration," and interpreted as the result of overwork. The next attack, if the patient is a woman, is often interpreted as the "melancholy of the menopause," by the man as due to his "business." The second point of importance in diagnosis, even of the mildest cases, is the **constancy of the early-morning unhappiness** which continues day after day for weeks, and which is little modified by any therapeutic measure, by change of environment, etc. Third, careful questioning will reveal the **pathological elements** in the patient's mental state: the lack of interest in life; the indifference to his family; the desire for death, etc.

One should exclude certain somewhat similar conditions: the "blues," which any emotional, overworked person may feel, but which rest in bed, a full diet, a change of scene, or good news will dispel; that depression which patients with long-continued eye strain may suffer during the period while presbyopia is developing, and which proper glasses will clear up; the depression which emotional persons suffering from some chronic, debilitating, and perhaps latent disease, as tuberculosis, arteriosclerosis, syphilis, or early cancer may feel, especially if it is their habit to worry about their health; and, finally, that depression which is an early phase of certain serious diseases, including general paralysis, dementia præcox, cerebral arteriosclerosis, etc.

The treatment of a case of manic-depressive psychosis is, first of all, an accurate diagnosis, and no pains should be spared to eliminate the several conditions mentioned above. During the attack the patient's **routine of life should be carefully organized.** If the condition is mild and is well explained to him he can coöperate in his treatment. It is better that the **patient be treated away from home** and from the scene of his business, preferably in some pleasant summer home, or in some quiet health resort or sanitarium. Nevertheless, if his case is very mild he may stay at home, and perhaps continue at light work. **He should never** be "isolated," nor, if married, should he **be separated from his wife** or she from

her husband. **He should never be alone. The early morning hours** are the most dangerous. Then even the mildest case may, in a brief moment of despair, commit suicide. **Definite fatigue should not be allowed,** therefore long walks, too much golf, strenuous gardening, and other forms of occupational therapy often increase the depression. No strenuous attempts should be made to amuse or entertain the patient, for even pleasant entertainment is fatiguing. **The diet** should be **regular and abundant,** regardless of the lack of appetite, for malnutrition only increases the severity of the condition. It is for this reason that the insane of this group are overfed even, if necessary, through a nasal tube. **The medicines** required are those to keep the bowels loose; mild hypnotics, if necessary, for sleep, but far better are warm packs and other hygienic measures; cod-liver oil; and others of symptomatic nature.

After the patient recovers from this attack he should then so organize his life that in the future too great physical or mental fatigue will not aid to precipitate another.

Dementia præcox is the diagnosis formerly made of about 15 per cent of the population of our insane hospitals. Now this percentage has risen to even 50 per cent or 60 per cent. The reason is not that this disease is more common than it formerly was, but because several conditions which formerly were classified under different names are now considered as forms of dementia præcox; catatonia, chronic mania, paranoia, some cases of depression, some of mania, hebephrenia, etc.

The essential point in dementia præcox is **the mental reduction which begins at about puberty,** although it may develop so slowly that it is not appreciated until middle life. **Children** with early dementia præcox are noticed by their teachers in school to be **a little odd;** their schoolmates, particularly, find them so and persecute them. They **tend to play alone.** They have a "shut-in" personality and the more their schoolmates torment them or the worse their case develops the more they **isolate themselves mentally** from their surroundings. Early, they are very apt to delight in reading

books far beyond their ability to comprehend; often they become very religious; they try their hand at invention, but without accomplishing much; they speculate in abstract philosophies; often they are too particular, too conscientious, or too neat; in fact, the "angel children" should always arouse suspicion. These patients early have peculiar feelings in the head, receive strange messages, see visions, or get peculiar ideas as to their bodies. For years these patients may continue to "live their own lives," and to earn their living, but they are rather moody, rather unstable, and often believe themselves to be misunderstood. Their periods of depression are often called attacks of "nervous prostration."

Sooner or later the more definite symptoms of dementia præcox appear; exhibitions of **temper without good cause**; a tendency to cruelty, and the patient quite at a loss to know why he is criticized or punished for his inhuman deeds; and some definite mental reduction. The patient may become very **paranoid,** that is, believe that those around him, although utter strangers, are plotting against his life or happiness, and may act accordingly, using violence and even homicide. This always should arouse suspicion. Periods of depression are common, during which **catatonia** may be a marked sign. That is, the patient, when left alone, sits still, not moving for hours, and will hold undisturbed for many minutes his hand or foot in whatever absurd position the examiner places it.

The prognosis of a case of dementia præcox which develops spontaneously is bad. The experience of the Great World War, however, showed that many young men who presented the typical clinical picture of this condition apparently did get well. For this reason some abandon the term "dementia præcox" for the less definite one, "schizophrenia."

Cases of dementia præcox **demand constant care.** They do best in institutions, not necessarily early in an asylum, but at least in an environment which lacks the emotional associations of the home. These are the cases which accumulate in our State hospitals. We try first to explain to the patient his

abnormal ideas and sensations and to plan for him a well-organized life with occupational therapy, physical therapy and a well-planned diet. In this way we attempt to reconstruct his life for him. Later, a hospital is the only safe place.

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